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USE OF SULFUR DIOXIDE IN TREATMENT OF THE EPIDEMIC COLD

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It is difficult to attack a problem which presents the confusion that is found in the question of the common cold. When the factor of etiology is so uncertain and indefinite, naturally the data concerning therapy and prevention will be even more unreliable. However, the subject is so extremely important when viewed from such angles as disability and complications that any scientific work along this line seems worthy of consideration.

It is not my intention to burden the reader by reviewing the extensive literature on this subject, as practically everything done along this line can be found in the large volume on the "Common Cold" by David and Robert Thomson [1] in the Annals of the Pickett-Thomson Research Laboratory.

In order to try out any form of intelligent treatment it is usually necessary to accept, temporarily at least, some theory as to the cause of the disease. At present the data on the causative factor in the common cold point to the following conclusions: 1. No definite organism has been established as the initiating agent. 2. Visible bacteria are probably secondary invaders and are concerned only in the severity and prolongation of the disease and its complications. 3. There is considerable evidence that colds are due to an invisible, unculturable, filtrable agent of some kind and that it is present only in the early stages of the disease. This view dates from Kruse's experiments and was confirmed more recently by Dochez in his work, with 44 per cent successful transmissions both in man and in apes.

While such factors as environment, allergy and anatomic defects undoubtedly play some rôle, I shall consider that the common cold is an infectious epidemic and endemic disease due to a filtrable virus.

From my experience it would seem that the period of incubation is from one and one-half to three days and the period of immunity is from three to four months. To avoid confusion in speaking of the

1. Thomson, D., and Thomson, R.: The Common Cold with Special Reference to the Part Played by Streptococci, Pneumococci, and Other Organisms, Ann. Pickett-Thomson Research Lab. **8**:1-699 (Dec.) 1932.

common cold, I am referring only to the epidemic disease that goes through families and communities at some periods of the year in the same manner as measles, whooping cough or any other communicable disease. I feel that many of the infections that are called colds are flare-ups of posterior sinusitis, with resulting pharyngitis and other symptoms similar to those occurring in the former condition. A patient often carries organisms in the posterior sinuses with some postnasal discharge, and these organisms suddenly become more virulent, or the patient's resistance becomes lowered, with a resultant flare-up, which is called a cold but which is not the same thing as the epidemic disease due to a filtrable virus. Also, I am not speaking of an epidemic streptococcic infection of the throat, which often travels up into the nose and accessory sinuses to produce similar symptoms.

In San Francisco there are generally two extensive epidemics of the common cold each year, one about the first of the year and the other in August or September. There are sometimes other smaller epidemics and also a number of sporadic cases.

In the case of practically all communicable diseases some persons are immune to infection while others are particularly susceptible to it. The common cold is no exception to this rule—many persons never have a cold, and another large group acquire this malady about as often as the infectious period will allow, that is, every three or four months.

Considering the vast amount of work done on the common cold, it is surprising that little or nothing has been reported on the blood count in this condition. Being unable to find any such data in the literature, in a recent epidemic of colds I had counts made in over thirty cases during the first twelve hours of the attack. The results in the majority of cases were essentially within normal limits. In three cases there was mild eosinophilia and in two cases slight leukocytosis. It was expected that the counts would show leukopenia, as in early influenza, but such was not the case.

In this same epidemic the temperatures during the first twelve hours were checked in over one hundred cases and found to be a few tenths of a degree above normal; rarely was there a temperature above 99.5 F., and rarely was there a subnormal temperature.

My brother, Dr. T. E. Rawlins, Assistant Professor of Plant Pathology at the University of California, who has been working on virus diseases of plants for a number of years, has found that filtrable viruses attacking plants, animals and man are similar in chemical and physical properties and that they do not behave toward ordinary antiseptics as do visible bacteria. For example, he noted that 70 per cent alcohol, a 1 : 1,000 solution of mercury bichloride or even a more concentrated solution, and 2 per cent hydrochloric acid have little effect in destroying these viruses. However, he found that low concentrations of sulfur dioxide (0.6 to 1.5 per cent) will readily destroy plant viruses

and the virus of foot and mouth disease. In the case of foot and mouth disease, 0.6 per cent sulfur dioxide was sufficient to destroy the action of the virus.

In discussing this subject with my brother one day, I conceived the idea that sulfur dioxide gas might likewise destroy the filtrable virus of the common cold if this antiseptic could be applied to the infected area in the early stage of the disease. It was found that this could easily be done by inhaling a concentrated solution of sulfurous acid, as this liquid gives off sulfur dioxide gas in large quantities. Sulfurous acid can readily be made by pouring sulfuric acid on sodium sulfate. Sulfur dioxide gas is liberated, and this can be bubbled into distilled water, producing in about two and one-half hours a saturated solution of sulfurous acid. The saturated solution is about 6 per cent.

In a recent winter epidemic treatment with sulfur dioxide was tried on over eighty patients, who were in the first stages of what appeared to be an epidemic cold. Of the eighty persons, fourteen received no relief and sixty-six stated that they were completely cured on the first or the second day after treatment. In nearly all these sixty-six cases, the patients reported that usually their colds lasted from one to two weeks, and they were practically all persons who were readily susceptible to the epidemic cold.

This same treatment was tried on other patients with colds during the third or fourth day of the attack, with practically negative results. In an epidemic of streptococcic throat this treatment likewise produced practically negative results.

In other words, it was found that in what was apparently an epidemic of colds sulfur dioxide gas inhaled in the first twelve hours of the disease stopped the cold in twenty-four hours in a large percentage of the cases.

There is little to be said as to the detail of the technic. The patient is given a 3 ounce (88.7 cc.) bottle half filled with a saturated solution of sulfur dioxide. By only half filling the bottle the fluid can be shaken, which tends to liberate more gas. The warmth of the hand holding the bottle likewise tends to liberate more gas. As this gas is volatile, a rubber stopper should be used. The patient is instructed to inhale the gas for about ten minutes three times during the first day. Deep inhalations are not necessary; the object is to get the disinfectant only into the upper respiratory tract. The ideal method is to fill the mouth and throat full without inhaling into the lungs and then, by closing the buccal cavities, to force the gas into the nasopharynx and the posterior part of the nose. Shallow inhalations may also be taken through the anterior nares.

The question as to whether or not sulfur dioxide is harmful to the patient is naturally important. This point has been rather thoroughly investigated by Drs. Kihoe, Machle, Kitzmuller and LeBlanc, of the department of physiology of the University of Cincinnati. They took up this problem from an industrial angle, since there are so many people employed in trades in which there is continuous or frequent exposure to sulfur dioxide gas. Two hundred men in a factory were studied; one hundred were workmen who were exposed to sulfur dioxide, and the other hundred performed work in a part of the factory where there were none of these fumes. The conclusion of the investigation was that there is no appreciable danger to health in a frequent or more or less continuous exposure to endurable concentrations of sulfur dioxide and that the effects of exposure to unendurable concentrations are negligible under conditions which allow quick escape.

I attempted to gain some information on this subject by sending questionnaires to employees who worked in sulfur dioxide fumes in a chemical plant, but unfortunately only ten replies were received; this was not enough to be of much help, but the average of these was about one cold in four years. The manager of one chemical plant recently told me that to his knowledge there were definitely few colds among men who worked in sulfur dioxide fumes. However, this information is so unreliable that it is of little value.

I am fully cognizant of the fact that from this comparatively small study I have no scientific proof of the value of sulfur dioxide in colds. In the first place, I have no definite diagnostic procedure for determining the presence of an epidemic cold in a patient. It cannot be verified with a Wassermann test as syphilis can, nor can the parasite be seen in the blood as in malaria. When a susceptible patient is seen during an epidemic of colds complaining of a cold and showing all the signs and symptoms of such disease, with a normal blood count and normal or slightly elevated temperature, it is naturally assumed that he has an epidemic cold. The number of patients treated is too small to be anything but suggestive of good possibilities. It would have been better to have treated every other patient by the recognized methods and every other one with sulfur dioxide and to have compared results.

However, I hope that some physicians will feel that the reason for using sulfur dioxide gas is sound and that the treatment is worth trying. I should be interested in hearing whether the results are positive or negative.

The following points are to be remembered in using sulfur dioxide in the treatment of colds.

1. It is beneficial only during the early stages of the infection. If this treatment is to be efficacious, patients must be educated to come for

treatment at the first signs of a cold, that is, a raw, dry, irritated feeling in the nasopharynx with general malaise, sneezing and other symptoms.

2. I have tried the 6 per cent U. S. P. solution of sulfurous acid, which can be procured at pharmacies, but this solution is far too weak and does not give off sufficient quantities of sulfur dioxide gas. The solution is probably old and, being volatile, has lost its strength by evaporation.

3. Sulfurous acid is a bleaching agent and should not be spilled on clothing.

4. Deep inhalations are not necessary and tend to make the patient cough, which discourages its use. Contact with the upper respiratory tract is all that is necessary.

A STATISTICAL STUDY OF ALLERGIC (VASOMOTOR) RHINITIS

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Sneezing, congestion of the nose and serous discharge without constitutional symptoms constitute a syndrome well known to all. Inspection of the nose shows little except intermittent turgescence of a pale mucous membrane. The condition is usually spoken of as some manner of rhinitis, catarrh or coryza. Its frequent occurrence in asthma and seasonal hay fever was recognized long before the word allergy was coined. In spite of its ubiquity it has never received an acceptable name. It is not an inflammation. Of the five classic symptoms of inflammation, "dolor, calor, rubor, tumor and functio laesa," only tumor and functio laesa are found, nor does the uncomplicated condition produce fibrosis in healing. In renal disease, a similar condition is distinguished from true nephritis by the term nephrosis. Were it not for the fear of confusion with rhinoceros, the term "rhinosis" might be suggested. However, with a mental reservation on the last four letters, the term allergic rhinitis is as good as any.

The condition is present to a greater or less degree in all those suffering from asthma and hay fever and complicates many true inflammations, both acute and chronic, of the nose and its sinuses. There is a history of nasal trouble preceding the onset of dyspnea by months or years in all persons with asthma. In the series about to be reported here, five persons acquired true asthma subsequent to the time they first consulted us and nine hay fever. The great majority, however, have escaped these more serious complications. About 50 per cent of hay fever sufferers have "catarrh" off and on during the winter. This close association with the frank manifestations of allergy caused one of us [1] to speak of the condition as "allergic respiratory disease" and to regard asthma and hay fever as complications of a fundamental process which, in its simplest form, is recognized as allergic rhinitis. This conception is furthered by roentgen examination of the nasal sinuses and of the lungs. This allergic swelling of the mucous membranes of the sinuses and bronchi produces a rather typical roentgen picture

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1. Clarke, J. A., Jr.: Pennsylvania M. J. **38**:408 (March) 1935.

and is seen in the lungs in most cases of hay fever even when there are no pulmonary symptoms.

The present study was made in an effort to learn something about allergic respiratory disease in its simplest form. In order to exclude from the series all persons suffering from asthma, all those with prominent cough, any dyspnea or even a mild "wheeze" were discarded. In many of these, the nasal symptoms were much more annoying than the mild troubles in the chest. Similarly, all those with a positive reaction of the skin to any pollen were excluded. These rigid requirements reduced the series to one hundred and sixty-two patients. These were taken from two thousand allergic persons studied in private practice. It should not be construed from this that allergic rhinitis constitutes only 8 per cent of clinical allergic conditions, because many of those discarded could hardly be classified otherwise. The study of this rigidly selected group showed some surprising differences between it and similar groups of asthma and hay fever sufferers.

This series consists of one hundred and sixty-two persons between the ages of 3½ and 66 years. On physical examination, they were a normal and attractive-appearing group. Thirty-seven per cent were referred by rhinologists, and another 42 per cent were referred by their family physicians. Seventy-three per cent had already had one and 20 per cent, two or more operations on the nose and throat. On the basis of family history, positive reactions of the skin, eosinophilia, typical roentgen findings and previous allergic manifestations in the patient himself, we were able to recognize the allergic diathesis in all but 27 per cent, irrespective of the nasal findings. We feel that there are few in the series for whom the diagnosis of allergic rhinitis is not justified.

The symptoms complained of were sneezing, blockage and discharge. Many came complaining only of frequent colds, and a fair percentage thought that they had seasonal hay fever. Seventy-seven per cent sneezed, 45 per cent had blockage, 43 per cent had nasal discharge and in 59 per cent the trouble was intermittent and described as "colds." Fifty per cent had eosinophilia. Fifty-eight per cent were females. Thirty per cent had a history of either hay fever or asthma in an immediate ancestor. Fifty-four per cent had had their symptoms less than five years, although 25 per cent placed their onset in childhood. Fifty-one per cent had positive reactions of the skin to one or more inhaled allergens, although in only 10 per cent was the reaction strongly positive. All tests were done by the intradermal method. Had the scratch test only been used, most of the weak reactions would have been missed, so that only a few more than the 10 per cent giving strongly positive reactions would have been considered cutaneously sensitive. From the clinical history and the response to treatment, we feel that

these weakly positive reactions are of great importance. When the intradermal reaction is suspicious, we are accustomed to use a subcutaneous reaction, injecting a fairly large dose beneath the skin. We regard a hot, red swelling, 1 inch (2.5 cm.) or more in diameter, as positive. We do not recognize clinical nasal susceptibility to an allergen in the absence of a positive reaction to that allergen, but we do recognize that the cutaneous reaction may be weak.

Since the present series excluded all those with reactions of the skin to pollens, house dust was the substance most frequently causing a reaction, producing positive reactions in 75 or 46 per cent, of the entire series. Feathers were next in frequency, with twenty-one persons reacting, and orris root (toilet powders) was third, with fourteen.

There were three persons reacting to cat, wheat flour and goat, two reacting to horse, insecticides, tobacco, buckwheat, cornmeal and dog and single persons responding to rabbit and kapok. There were

Summary of Data Concerning Persons with Allergic Respiratory Diseases

	Allergic Rhinitis, Percentage	Asthma, Percentage	Seasonal Hay Fever, Percentage
Family history of allergy	30	50	50
Scarlet fever	30	20	12
Females	58	42	43
Strongly positive reactions of the skin	10	52	100
Moderately positive reactions of the skin	41	22	..

only seven reactions to food. None of the reactions to a food was duplicated. The list included rice, lima bean, spinach, crab, potato, asparagus and oats. We are unable to agree with those who place a great deal of importance on ingestive contact. We were unable to find any one in whom the sole cause of trouble was a food or a group of foods. Food testing has not been a part of our routine for many years. All patients observed in the early period were tested with foods, and in the later years those who did not respond satisfactorily were given food tests. The results did not justify the effort.

Psychiatrists have, for the most part, recognized that asthma and hay fever are no longer to be grouped with the neuroses. Under the name vasomotor rhinitis, however, they are still inclined to consider the present subject as in their province. It is true that the first stage of weeping or crying is similar to allergic catarrh. While we did not psychoanalyze our patients, this thought was kept in mind, but in only eight persons in the series did we feel that unusual psychic conditions were present. Of these eight, five were women and three were men. The ages of this group varied from 15 to 50, with an average of 37. Three had positive reactions of the skin, and adequate treatment was

given to five. An excellent result was obtained in two, while in only one did the treatment fail completely. These figures are practically the same as we found in the entire series. It is our belief that psychic influences have little to do with the condition, except that the discomfort of allergic coryza plus the statement of a physician that the trouble is "nerves" is likely to upset an otherwise normal psychic balance.

Treatment followed the usual allergic routine. Substances giving a positive reaction of the skin should be removed as completely as possible. At times this is most simple—a dog or a cat. Except in rare instances, it is possible completely to remove from a person's environment all substances except house dust and orris root. When the reaction to these is positive, it is usually necessary to give a course of hypodermic injections of a solution of these substances. These injections are given at weekly intervals, each dose being greater than the preceding one until complete clinical relief is obtained. After this, injections are continued at intervals of one month for one year after all symptoms have ceased. It must be remembered that the injections are not a substitute for elimination but are given in addition to elimination. Injections of allergens prepared from dust without proper attention to the home are usually of little benefit. Likewise, injections of allergen prepared from orris root will fail if the woman insists on using toilet powders containing this substance. One should never give injections of allergens prepared from a dog with the idea that they will obviate the necessity of removing the dog.

House dust is so important that it deserves a paragraph to itself. Years ago, Cooke,[2] discovered that it is possible to obtain strongly positive reactions of the skin to sweepings (or to the dust obtained from a vacuum cleaner) in persons in whom it is impossible to get a positive reaction with any other substance. The accuracy of this observation has been proved uniformly by all workers in the field. While no secret was made of this discovery, the finding was not published for years because the discoverer felt that he must find the origin of the active principle in dust. Many others have spent much time and money in a vain effort to solve what at first appears to be an easy problem. Furthermore, this active principle is not the same in all dusts. One person will react to the dust of one house and another will not but will react to another sample. We believe that there are five or six different substances contained in our stock preparation of dust, which is made from samples of dust from different houses. That it does not come from any substance deliberately introduced into the house (as in furniture) has been proved. Yeasts and molds have been suspected but have been proved to be allergic in only one instance. Recently,

2. Cooke, R. A.: J. Immunol. **7**:147, 1922.

Cohen [3] of Cleveland has advanced some interesting facts pointing to some subtle change in cotton linters, which is used as stuffing in mattresses, upholstery or children's toys. As the problem stands at present the dust from almost all private residences contains some harmful substances to a greater or less degree. In hospitals, modern apartment houses and business buildings dust is not a factor. Much can be done to reduce the inhalation of this substance by the sufferer. The bedroom should receive the greatest attention. Carpets should be removed and likewise upholstered furniture and heavy draperies. A thorough cleaning of the bedroom should take place twice a month rather than the customary twice a year. Attics, cellars and storerooms should be kept clean. Waterproof coverings for mattress and pillows are of value but should not replace frequent cleanings. Only when the patient cooperates and makes these changes do we get satisfactory results from injections of allergens prepared from dust.

In discussing removal of foci of infection, we wish to call particular attention to painless abscesses at the roots of the teeth. Physicians are too prone to accept a set of teeth without cavities as normal. Roentgenograms and tests for vitality are desirable in all cases. We have seen frank abscesses in persons in the early twenties. In cases of stubborn allergic rhinitis we have all dead teeth removed regardless of negative roentgen findings. It should also be remembered that a fragment of a tooth may be the seat of an abscess, even with a normal gum above it.

Diseased tonsils, of course, should be removed, and frank infection of the nasal sinuses should receive appropriate treatment. There are some persons who become asthmatic after a tonsillectomy or some nasal operation. A careful history will usually show that the operation was done because the symptoms were becoming rapidly worse just prior to the operation, so that something had to be done and the tonsils came out. We doubt a causal relation between the operation and the asthma. However, it is often difficult to convince the patient. For this reason, we prefer to delay operation until after two months of allergic treatment. This, of course, does not mean that obstructive polyps should not be removed or an infected antrum drained. But the surgical procedure should be essentially palliative until the patient is properly prepared. We believe it inadvisable to operate on any allergic nose until a competent allergic study has been made.

To those with frank infection and those with negative reactions of the skin we give stock vaccine and occasionally autogenous vaccine. We feel that autogenous vaccines should always be fortified with stock vaccine because of the probability of additional bacteria entering the

3. Cohen, M. B.: J. Allergy **6**:517, 1935.

nose. Vaccine should not be given too frequently or in too large doses. Large local reactions are not desirable and often aggravate the condition. The injections should be started at weekly intervals and later continued at monthly intervals for an indefinite period. It should be remembered that injections of vaccine are not a substitute for specific therapy when this is indicated by a positive reaction of the skin. On the other hand, a little vaccine added to specific treatment is frequently a great help.

The results are based on a simple questionnaire sent out in November 1935. In most instances this was years after treatment was started. We asked whether the patient had followed our advice and if so, whether the condition was improved. We discarded all who did not take treatment for at least two months. The results were surprisingly good: 23 per cent were entirely relieved and 56 per cent decidedly improved, giving 79 per cent satisfactory results.

Returning to a comparison with asthma and hay fever, the most striking difference is the sex distribution, which has been observed by other writers.[4] In a similar series of patients with asthma and hay fever from the same practice, males predominate, while among patients with allergic rhinitis females are more numerous—for asthma and hay fever, 43 per cent females; for allergic rhinitis, 58 per cent females. The possibility that "cry baby" women, not at all allergic, were included in the series is not borne out by a detailed study. There were more women than men with eosinophilia. Of those with negative reactions of the skin, 60 per cent were women, which compares with the 58 per cent for the entire series. The percentage of failures was as great in the females as in the males, although excellent results occurred almost three times as frequently in women. We ascribe this to more faithful treatment. It is possible that some hormone peculiar to females enables them to escape the more serious forms of allergy, but it certainly is of no benefit to them after the development of either asthma or hay fever.

One of us (J. A. C.) noted in an unpublished communication to the section of medicine of the College of Physicians of Philadelphia that scarlet fever occurs almost twice as frequently among persons with hay fever as among those with asthma. In our present group of persons with allergic rhinitis, 30 per cent had had scarlet fever, compared to 20 per cent of patients with hay fever and 12 per cent of patients with asthma.

A family history of uncomplicated allergic rhinitis is unreliable because the condition is too trivial to be remembered except in the

4. Bray, G. W.: Recent Advances in Allergy, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1934, p. 314.

immediate family. For this reason, we asked only for asthma and hay fever. These diseases occurred in the families of 30 per cent of the present series, as compared with an incidence of only 50 per cent in the families of those suffering from asthma and hay fever. We believe that the percentage of family histories of allergic conditions would be as great as for persons with asthma and hay fever were it as easy to get an accurate account of sneezing attacks as it is to get the history of asthma.

The poor reactivity of the skin seems to be a real characteristic. Hay fever, of course, gives 100 per cent strongly positive reactions of the skin. Asthma gives 75 per cent positive reactions, of which 52 per cent are marked. In persons with allergic rhinitis, we obtained only 51 per cent positive reactions, and only 10 per cent of these were marked.

The good results compare with those obtained in hay fever, both conditions yielding to treatment more easily than does asthma.

If we may be permitted a moment of speculation, we regard allergic rhinitis as the initial and simplest form of a general allergic disease of the respiratory tract, occurring equally in the two sexes. For some unknown reason, men are more likely to have the more serious forms of the disease (asthma and hay fever), thus giving the disproportion of the sexes noted in the present study. Similarly, scarlet fever seems to exert a benign influence in preventing the development of asthma.

CONCLUSIONS

Allergic rhinitis is the simplest form of allergic disease of the respiratory tract. It frequently precedes the development of true asthma and hay fever.

It differs from asthma and hay fever in a preponderance of females, in a greater susceptibility to scarlet fever and in weaker cutaneous reactions.

It responds well to allergic treatment.

PATHOLOGIC CHANGE IN OLFACTORY NASAL MUCOSA OF ALBINO RATS WITH "STUNTED" OLFACTORY BULBS

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The occasional occurrence of small olfactory bulbs in the albino rat has been noted by Holt,[1] Sugita[2] and Donaldson and Hatai[3] and in my previous study.[4] These bulbs, which occur rather infrequently in young rats and which were designated as stunted by Sugita, were found to be present in all the senile rats studied.[4] The fact that the decrease in volume was apparently dependent on the loss of the entering olfactory fibers led me to obtain some further material in order that the olfactory mucous membrane and olfactory nerves might be studied along with the olfactory bulbs. The results of this study form the basis of the present communication.

MATERIAL AND METHODS

Twelve male albino rats of the experimental colony strain were obtained from the Wistar Institute for this investigation. These were of the same stock as those used in a previous study and like them were all normal rats raised under standard conditions. It should be mentioned, however, that in this case the animals were not killed at the Wistar Institute but were shipped alive to Toronto and the required parts removed and prepared there. To reduce the period of change from standard conditions to a minimum the animals were killed immediately on arrival, that is, within twenty-four hours of their leaving the colony.

From the Department of Biology, University of Toronto.

This study was made possible by a grant from the Trustees of the Banting Research Foundation, and was carried out under the direction of Dr. E. Horner Craigie.

1. Holt, C. M.: Studies on the Olfactory Bulbs of the Albino Rat: I. Experiments to Determine the Effect of a Defective Diet and of Exercise on the Weight of the Olfactory Bulb; II. Number of Cells in the Bulb, J. Comp. Neurol. **27**:201-261, 1917.

2. Sugita, Naoki: Comparative Studies on the Growth of the Cerebral Cortex: I. On the Changes in Size and Shape of the Cerebrum During Postnatal Growth of the Brain; Albino Rat, J. Comp. Neurol. **28**:495-510, 1917.

3. Donaldson, H. H., and Hatai, S.: On the Weight of the Parts of the Brain and on the Percentage of Water in Them According to Brain Weight and Age, in Albino and in Wild Norway Rats, J. Comp. Neurol. **53**:263-307, 1931.

4. Smith, C. G.: The Change in Volume of the Olfactory and Accessory Olfactory Bulbs of the Albino Rat During Postnatal Life, J. Comp. Neurol. **61**: 477-508, 1935.

The animal was chloroformed, and Zenker's solution at 37 C. was injected into the head through the arch of the aorta. The skull was then fixed in the same solution for twenty-four hours, washed for twenty-four hours in running water, treated with 70 per cent alcohol for three days, decalcified in a 5 per cent aqueous solution of nitric acid and washed in water for twenty-four hours. The tissue was then dehydrated and embedded in paraffin. Serial sections 10 microns thick were prepared, and every tenth section was mounted and stained with hematoxylin and eosin.

OBSERVATIONS

The Olfactory Bulbs and the Olfactory and Vomeronasal Mucous Membrane.— The olfactory bulbs of all twelve animals were examined for stunting, and the results are given in the accompanying table. In the first column is given the number by which each rat was designated. Rats 87, 88 and 89 were 3 months old, and the others ranged between

The Age of the Animals and the Condition of the Olfactory, Vomeronasal and Nonolfactory Mucous Membranes on the Right and the Left Side *

Rat	Age, Days	Condition of Olfactory Bulb		Condition of Olfactory Mucous Membrane		Condition of Vomeronasal Organ Mucous Membrane		Condition of Nonolfactory Mucous Membrane	
		Right	Left	Right	Left	Right	Left	Right	Left
87	92	N	S	N	D	N	N	N	D
88	92	N	N	N	N	N	N	N	N
89	92	S	S	D	D	N	N	D	D
90	890	S	S	D	D	N	N	D	D
91	770	N	N	N	N	N	N	N	N
92	992	N	S	N	D	N	N	D	D
93	912	S	S	D	D	D	D	D	D
94	858	S	N	D	N	N	N	D	D
95	995	S	S	D	D	N	N	D	D
96	857	N	N	N	N	N	N	N	N
97	872	N	N	N	N	N	N	N	N
98	778	S	S	D	D	N	N	D	D

* N indicates normal, S stunted and D diseased.

2 and 3 years of age. In column 3 the character of the right and the left bulb for each animal is shown, S designating a stunted bulb and N a normal one. Among the young rats, there was one with normal bulbs on both sides, one with stunted bulbs on both sides and one with a normal bulb on one side and a stunted bulb on the other. Of the older rats there were three with both bulbs normal, four with both bulbs stunted, one with the right bulb stunted and the left bulb normal and another with the right bulb normal and the left stunted. There had been no attempt at selection of the rats; all were supposed to be in perfect condition.

The olfactory mucosa and the olfactory nerves of each half of the nose were examined, and it was found that without exception wherever a stunted bulb occurred the olfactory mucosa was diseased, the olfactory cells were wanting and the olfactory nerves had atrophied. This finding is particularly interesting when it is considered that when a stunted bulb occurred on only one side it meant that one half of the

nasal cavity was diseased and the other had remained normal. In column 4 these observations are tabulated, N designating a normal mucosa and D a diseased one.

The table also gives information regarding the condition of the vomeronasal organ. This was investigated particularly because it had previously been shown that the vomeronasal center showed no abnormality even in bulbs in which the olfactory center was stunted. As the table shows, the organ was normal in every animal except rat 93, in which both sides were diseased. The fact that it was involved in this animal and in none of the others is explainable by the observation that in this

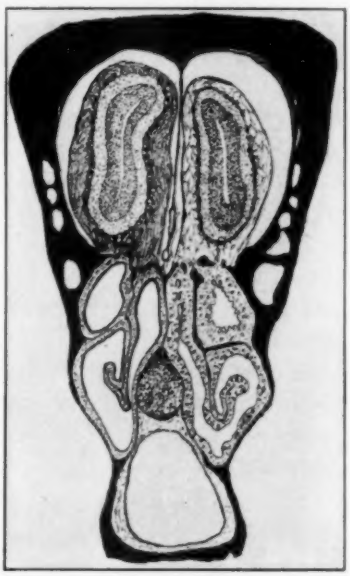


Fig. 1.—Cross-section through the posterior part of the nose. The left olfactory bulb is normal, the right stunted. The drawing was made from a section magnified 5 diameters.

case the diseased condition had progressed to the stage at which there was a complete disorganization of the nasal cavity. The vomeronasal center as well as the olfactory center was stunted in this animal.

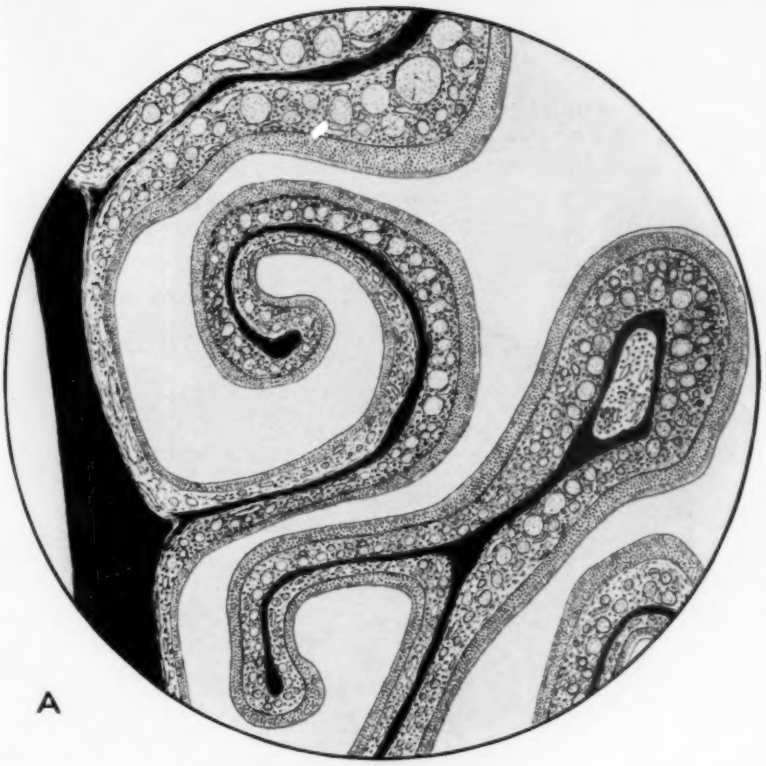
In order to illustrate the characteristics of the pathologic condition, figures 1 to 8 have been prepared. Figure 1 shows a cross-section of the posterior part of the nose. The section is outlined by the bone of the skull. Within are the olfactory bulbs separated from the nasal cavity by the cribriform plate. The septum divides the nasal cavity completely at this level and abuts on the nasopharynx below. This section was taken from rat 94, in which a stunted and a normal bulb were present

in the same subject. The normal bulb is shown on the left, with the masses of the olfactory nerve fibers streaming through the cribriform plate and entering it. The nasal cavity on the same side shows a perfectly normal mucous membrane, the characteristics of which will be shown later. On the right side is shown the stunted bulb, which is small. The olfactory nerves are wanting here, leaving an open meshwork of connective tissue within which the olfactory fibers had previously found their way to the brain. The nasal cavity of this side shows a diseased condition of the mucous membrane. It is swollen, with an irregular surface, over which the olfactory epithelium, even with this magnification, can be seen to be wanting. It is of interest that the signs of inflammation—edema and infiltration with wandering cells—are limited to the nasal cavity by the cribriform plate. All that is seen in the cranial cavity is the atrophy of the olfactory nerves.

In figure 2 *A* is shown a portion of the nasal cavity containing the ethmoid turbinates. Here are shown the normal sensory olfactory epithelium (to be described later) and in the tunica propria the large bundles of the olfactory nerves which pass to the olfactory bulb. This figure is introduced to permit the comparison of the normal with the pathologic condition shown in figure 2 *B*, in which a similar field has been selected from a rat with a stunted bulb. Three features are strikingly shown in this drawing: (1) the loss of the normal olfactory epithelium, (2) the disappearance of the olfactory nerves from the tunica propria and (3) the presence of adhesions, which are a frequent finding in the nasal cavities with this abnormality.

The aforementioned sections have illustrated the evident relationship between the loss of the olfactory nerves and the presence of a diseased mucosa. The detailed characteristics of the change in the olfactory epithelium and the tunica propria are best shown in the series of drawings of highly magnified specimens. Figure 3 *A* shows the normal olfactory mucous membrane. There are three kinds of cells in the epithelium, the basal cells, the supporting cells and the sensory olfactory cells. The basal cells are cone-shaped elements with large vesicular nuclei. The bases of these cells lie next to the tunica propria. The supporting cells are tall columnar elements lying in regular arrangement side by side at the free margin of the epithelium. Proximally these cells send delicate strands of cytoplasm downward to be anchored among the basal units at the deep edge of the epithelium. Distally at the free border are delicate processes which are not, however, true cilia (Kolmer [5]). Enmeshed in the anchoring cytoplasmic strands of the

5. Kolmer, Walter: Geruchsorgan, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 3, pp. 192-249.



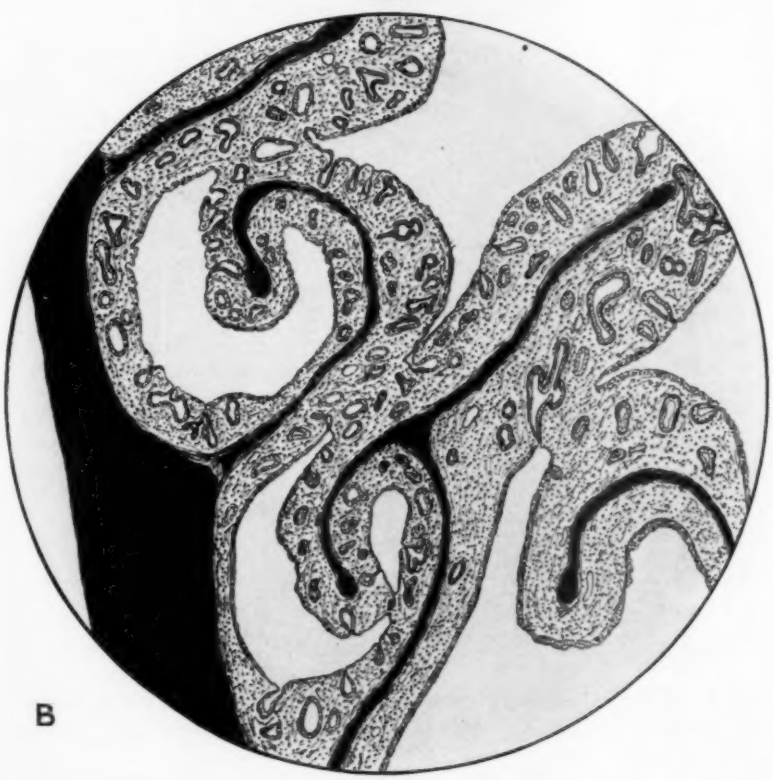


Fig. 2.—Sections from the ethmoid turbinates, showing (*A*) the normal olfactory mucous membrane and (*B*) the character of the olfactory mucous membrane when the olfactory bulbs are stunted. These drawings were made from sections magnified 60 diameters.

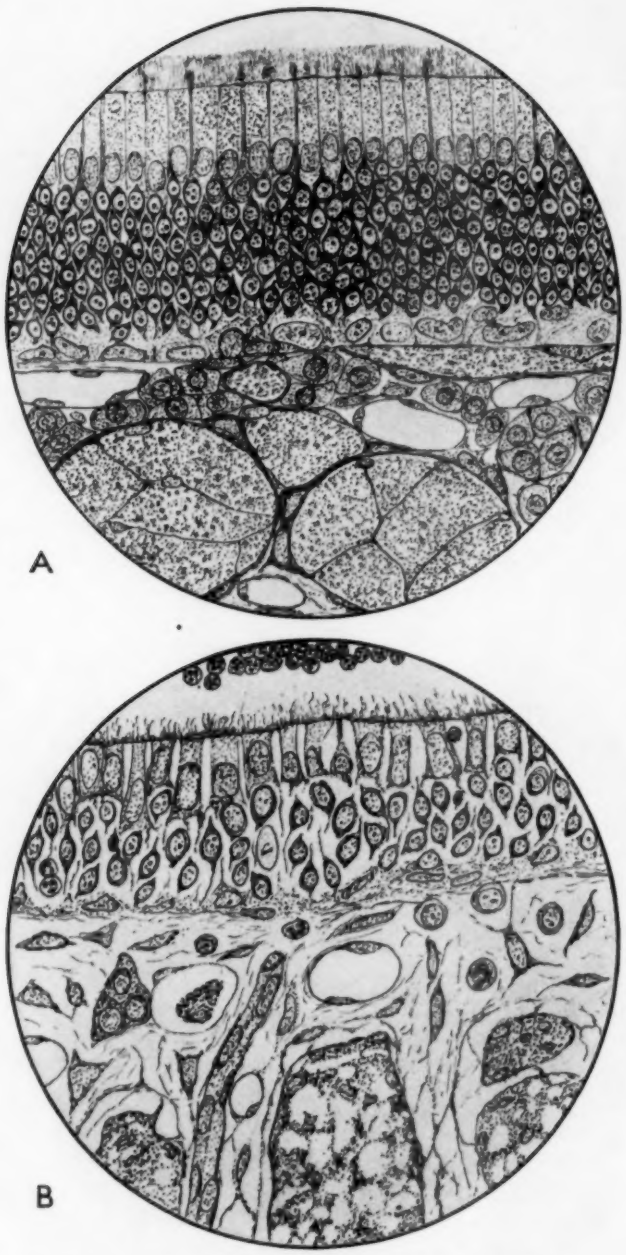


Fig. 3.—*A*, normal olfactory mucous membrane (× 600); *B*, olfactory mucous membrane showing beginning destruction of sensory cells and degeneration of olfactory fibers (× 600).

supporting elements are the sensory olfactory cells. These are fusiform, and each has a proximal and a distal process. The distal process passes up between the supporting cells to end in a small olfactory vesicle which lies above the cuticle of the epithelium, and from this arise the olfactory hairs (Kolmer[6]). The proximal processes of these cells pass toward the deep edge of the epithelium and into the tunica propria where, joining with other fibers, they form bundles of olfactory nerves as shown. To be seen also in this figure within the tunica propria are the glands of Bowman, which are characteristic of the olfactory area.

Figure 3 *B* illustrates an early stage in the development of the pathologic condition in the mucosa. In this specimen the elements of the epithelium are separated by edema and there is a considerable reduction in the number of the olfactory cells. In the tunica propria there is also considerable edema, with evidence of degeneration in the olfactory nerve bundles, which is apparently related to the loss of sensory elements in the epithelium.

Figure 4 *A* represents a stage at which all sensory olfactory cells and all olfactory nerve fibers have disappeared. Here is shown a condition which is dependent on regeneration of the nonsensory cells of the epithelium. As evidence of this one frequently finds mitotic figures in what appear to be the remaining supporting, or basal cells, and in the cells of the ducts of Bowman's glands. The result of this attempt at repair in the presence of a continued process of exudation gives rise to the picture as shown in this figure, in which the epithelium is represented by a loose meshwork of cells that have not differentiated into the characteristic columnar or cuboidal epithelial elements.

When active destruction occurs, the whole of the epithelium may be destroyed, leaving a denuded tunica propria. In this case, the continuity of the epithelium may be restored locally by an extension of cells from the ducts of Bowman's glands in which active mitotic division is occurring. Figure 4 *B* illustrates such a case.

In those instances in which healing is permitted to occur, differentiation of these cells proceeds, giving rise to tall ciliated columnar epithelium as shown in figure 5. Here no evidence of regeneration of olfactory cells is to be seen, and in the tunica propria no evidence of olfactory fibers is to be found.

These sections illustrate the manner in which the olfactory cells are destroyed, leading to an associated loss of olfactory fibers, which I have suggested to be the factor responsible for the stunting of the olfactory bulbs.[4]

6. Kolmer,[5] p. 198.

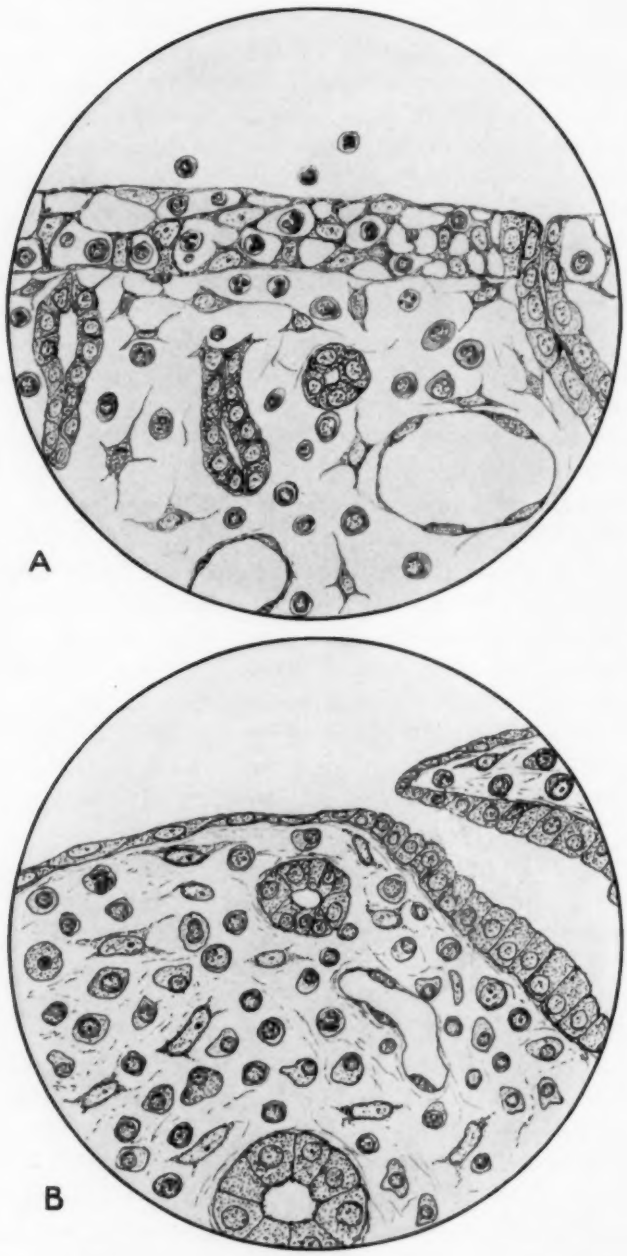


Fig. 4.—*A*, olfactory mucous membrane showing absence of sensory cells and olfactory nerves (× 600); *B*, olfactory mucous membrane showing early stage in repair (× 600).

Although, as just described, there does occur localized repair in the olfactory epithelium, in no case was it found proceeding toward completion. It would seem that the condition is a fluctuating one, an area now healing, now suffering a reinvolvement, so that after a time the nasal mucosa becomes much thickened and numerous adhesions develop between contiguous epithelial surfaces. This may reach a stage at which, as in rat 93, it is difficult to imagine that a passage for air exists.

The characteristics of the pathologic change in the vomeronasal organ, as judged from the single example found in rat 93, are essen-

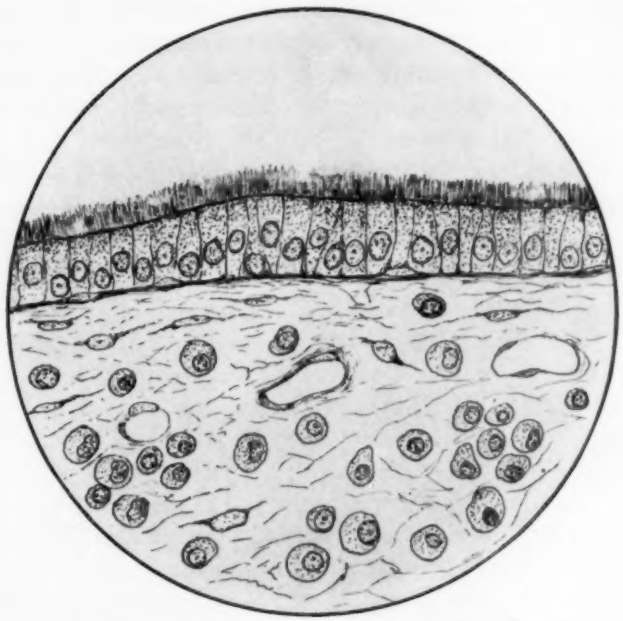


Fig. 5.—Olfactory mucous membrane showing replacement of sensory area by ciliated columnar epithelium (× 600).

tially those described for the olfactory mucous membrane. There are a loss of sensory cells and a proliferation of the remaining supporting elements which tend to differentiate into the columnar ciliated cells.

The Nonolfactory Portion of the Nasal Cavity.—The anterior part of the nose also shows evidence of disease. This does not involve the region of the external nares, which is covered by squamous epithelium, but where the latter passes over into the columnar epithelium via a transitional zone in which the cells are more or less cuboidal and in which the epithelium is about 4 to 6 cells in thickness, edema and infiltration with inflammatory cells begin to be evident. A marked thickening of the mucous membrane with considerable deep folding of the

epithelium may occur. The cells lining the infolded pockets take on a columnar shape and may even simulate goblet cells. As the region of ciliated columnar epithelium is attained, one finds in cases of less severe involvement only an increase in the height of the epithelial cells, with a slight infiltration of the tunica propria with round cells. When the reaction is more intense, one finds that both the tunica propria and the epithelium are heavily infiltrated with inflammatory cells, leading to a marked increase in the thickness of the mucosa and a definite tendency to folding. Considerable inflammatory exudate consisting of polymorphonuclear leukocytes is to be found in the nasal cavity.

The table shows that the mucous membrane of the anterior part of the nose is diseased in every case in which the olfactory mucous membrane is involved. However, in one animal, rat 94, the former was found to be markedly diseased up to the beginning of the olfactory epithelium, while the latter was normal.

Other Pathologic Changes.—The maxillary sinus was involved in all cases in which the nasal cavity was diseased. Here, except for the absence of a tendency for folds to develop in the epithelium, the same types of reaction were found as in the portions of the nasal cavity lined by ciliated columnar epithelium.

The middle ear was found to contain macroscopic pus in rats 89, 95 and 98. In rat 95 there was also a huge abscess of the brain, involving the whole of the temporal lobe of the cerebrum.

The lungs of all twelve rats showed evidence of disease in which there was a cellular infiltration of the bronchial walls with partial or complete occlusion. This was not as marked in the young as in the older rats. Distally the bronchi were distended in some animals with mucoid material containing large numbers of polymorphonuclear leukocytes, and the lung either was collapsed or showed distention of the alveoli by this same material. In one case an adenomatous nodule was found, and in several others solid nodules of hyperplastic epithelial tissue were encountered.

COMMENT

A reference to the marked variation in size of the olfactory bulb has appeared in the report of every investigator who has attempted quantitative studies on this part of the brain. (It so happens that all have worked on rats of the Wistar stock.) In a previous study, in which the volume of the olfactory center was measured, it was found that a variation up to 25 per cent may occur without change in structure but that beyond this there is evidence of a loss of olfactory nerve fibers. In observations reported here it is clear that the loss of olfactory nerves is due to a destruction of sensory olfactory cells in the mucous membrane of the nose as a result of disease. The loss in volume of

the bulb, however, is not due alone to the removal of the olfactory fibers but is partly also the result of an atrophy of neurons in the bulb. This was shown by making counts of the mitral cells with which the olfactory fibers synapse. It was found that there was a decrease from approximately 70,000 in the normal to about 40,000 in the stunted bulb. If this conception of the development of small bulbs is accepted, the term stunted should be replaced by atrophic.

The fact that atrophic olfactory bulbs were found in all the senile rats [4] would suggest, if other conditions are taken to be constant, that these animals, owing to their advanced age, had a lessened resistance to the disease process described, so that all were suffering from it.

In the light of these findings it becomes of interest to consider the stunting effect of malnutrition during active growth, as reported by Holt.[1] Plates 1 and 2 in her paper suggest that a decrease in the thickness of the outer layer of fibers had occurred similar to that which I have shown is to be expected in bulbs in which stunting is due to a diseased condition of the olfactory mucous membrane. On the other hand, in plate 3 it is probable that both the control and the test bulb were from rats with normal olfactory mucous membranes. The stunting in this case is not of the type with which this study is concerned but is an example of arrested development. The fact that she obtained relatively small olfactory bulbs in these animals when comparing them with controls of the same age is not surprising since, as shown in figure 21 of my earlier paper,[4] the relative volume of the bulbs normally increases rapidly during this particular period. In addition to this, however, when one considers the weakened condition of the underfed animals, which she states was marked, it would seem not unlikely that these animals should contract the nasal disease described here and in this way secondarily acquire stunted olfactory bulbs. As a possible argument against this it may be suggested that she did not find a reduction of the number of mitral cells to below 70,000. It must be remembered, however, that her experiments lasted only from thirty to sixty days, so that the secondary atrophy of the mitral cells might not yet have occurred. My findings suggest that in all probability the stunted bulbs which she noted in the sick rats were the result of an associated rhinitis.

A replacement of the olfactory epithelium by nonsensory ciliated respiratory epithelium in man as the result of disease was described by Suchannek [7] and by Kolmer.[8] Suchannek also pointed out that one

7. Suchannek, Hermann: Differentialdiagnostische Merkmale zur Unterscheidung zwischen normalem und pathologischem menschlichen Riechepithel, resp. respiratorischem Flimmerepithel, Ztschr. f. Ohrenh. **22**:4-10, 1892.

8. Kolmer,[5] p. 203.

may encounter stratified squamous epithelium. Bauer and Beck[9] in figure 29 in their atlas showed stratified epithelium replacing olfactory epithelium. Kolmer stated that the sensory olfactory epithelium is replaced by an ingrowth of ciliated epithelium from the adjacent respiratory region. There was no mention of the ciliated columnar cells arising from the proliferation of undestroyed supporting cells or from the cells lining the ducts of Bowman's glands, as I have described them in the rat. The findings of the aforementioned workers would indicate that in man, as in the rat, destruction of the olfactory cells and hence of the olfactory nerves may occur.

In an endeavor to determine the nature of the condition which was evidently the cause of the stunted bulbs in my material, the work of Rudolf Jaffé[10] and that of Oskar Seifried[11] were consulted. In these no specific reference was made to the rat in the description of diseases of the nose, but it was found that the symptoms, signs and pathologic findings in the disease of rabbits called rhinitis contagiosa cuniculorum were similar to those shown by the rats of my study. This disease, according to these authors, is an infectious one characterized by languor, loss of appetite and frequent sneezing, with considerable nasal discharge. The animals rub their noses repeatedly with their forepaws. The course of the disease may be acute, subacute or chronic. Almost all those with acute conditions die, and among those with chronic involvement complete recovery is seldom observed. Frequently, according to Seifried, the manifestations of the disease may be so slight that they are completely missed. The condition is characterized pathologically by a more or less severe inflammation of the nasal and pharyngeal mucous membranes and later of the trachea and lungs. As complications, suppuration of the middle ear, meningitis and abscesses of the brain are found. The disease is described as widespread. According to Webster,[12] the incidence in the stock of the Rockefeller Institute varied from 60 per cent in September and October to 20 per cent in the early summer months.

From the description, although no statement is made regarding whether it does or does not involve rats or other animals in addition to rabbits, it would seem not unlikely that this is the disease from which the animals were suffering.

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9. Bauer, Theodor, and Beck, Oscar: Atlas der Histopathologie der Nase und ihrer Nebenhöhlen, Leipzig, Curt Kabitzsch, 1924.

10. Jaffé, Rudolf: Anatomie und Pathologie der Spontanerkrankungen der kleinen Laboratoriumstiere, Kaninchen, Meerschweinchen, Ratte, Maus, Berlin, Julius Springer, 1931.

11. Seifried, Oskar: Die wichtigsten Krankheiten des Kaninchens, mit besonderer Berücksichtigung der Infektions- und Invasions-Krankheiten, Munich, J. F. Bergmann, 1927.

12. Webster, cited by Seifried.[11]

SUMMARY

So-called stunted olfactory bulbs are not an exaggeration of a normal variation in size but are a secondary manifestation of a chronic disease. The failure to recognize this previously is explained by the fact that the primary manifestations of the pathologic condition may be so slight as to be entirely missed.

The disease is characterized by a more or less severe inflammation of the olfactory mucous membrane, which may be limited to a part or all of one side of the nose or may involve both sides. The olfactory sensory cells are destroyed causing atrophy of the olfactory nerve fibers and, secondarily, atrophy of the olfactory bulbs.

The healing of the olfactory mucous membrane involves a replacement by ciliated columnar epithelium, which apparently arises from the undestroyed supporting and basal cells and the cells of the ducts of Bowman's glands.

My observations indicate that the vomeronasal organ is rarely involved in the pathologic change, a fact which appears to explain why the vomeronasal center is not so subject as the olfactory center to the atrophic changes described.

It is suggested that the disease process is similar to that found in rabbits and called rhinitis contagiosa cuniculorum.

COMPLETE APICECTOMY (MASTOIDOTYMPANO-APICECTOMY)

A NEW TECHNIC FOR THE COMPLETE EXENTERATION OF THE APICAL CAROTID PORTION OF THE PETROUS PYRAMID

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NEW YORK

In 1936 I[1] published a preliminary report on a new technic for complete apicectomy. I desire here to establish the rationale of this technic and also to discuss the compelling requirements for such a procedure. The time has now arrived when surgical exenteration of the pathologic focus should replace drainage of the accumulated pus and be the goal of surgical intervention.

Petrositis is an inflammation of the petrous portion of the temporal bone, as mastoiditis is an inflammation of the mastoid portion of this bone, but, unlike the definitive term mastoiditis, the term petrositis is too broad and vague. From a surgical point of view the petrous pyramid differs from the mastoid process in that the former is crowded with vitally important anatomic structures. The term petrositis is meaningless to the otologist if he is to intervene surgically. In mastoiditis, even though the suppurative process may involve a small portion only of the bony structure, surgical intervention necessitates destruction and eradication of the whole mastoid process. On the contrary, in an infection of a portion of the petrous pyramid, it is necessary to localize the area infected, so that the adjacent vitally functioning structures are not destroyed. The nomenclature of suppuration within the petrous pyramid should conform to and be descriptive of the anatomic structures within the petrous pyramid involved by the pathologic process. Suppuration within the petrous pyramid may result in the following conditions:

1. Suppurative labyrinthitis.—Suppuration involving the membranous labyrinthine structure proper
2. Suppurative perilabyrinthitis.—Pathologic involvement of the perilabyrinthine cell groups surrounding the labyrinth without involvement of the labyrinthine structure proper; when fistulas accompany this pathologic condition, they may be located in:
   (*a*) The posterior fossa wall of the basal labyrinthine portion, anterior to the posterior semicircular canal, forming an epidural abscess

1. Lempert, J.: Complete Apicectomy: Preliminary Report of a New Technic, New York State J. Med. **36**:1210 (Sept. 1) 1936.

(*b*) The superior middle fossa wall of the basal labyrinthine portion, anterior and internal to the eminentia arcuata, forming an epidural abscess

(*c*) The anterior tympanic wall of the basal labyrinthine portion in the supracochlear region

(*d*) The anterior tympanic wall of the basal labyrinthine portion in the infracochlear region

3. Suppurative apicitis.—Suppuration of the cellular area within the apical carotid portion of the petrous pyramid with or without fistulas; fistulas may exist in:

(*a*) The anterosuperior wall between the cochlea and the bend of the internal carotid canal

(*b*) Any part of the external bony wall of the transverse portion of the carotid canal, draining a pericarotid abscess

(*c*) The posterior fossa wall anterior to the internal auditory meatus forming an epidural abscess

(*d*) The inferior skull base surface

4. A combination of suppurative apicitis and suppurative perilabyrinthitis

Every one of these pathologic conditions presents a different clinical picture and requires direct and specific surgical treatment of the special anatomic structure involved without sacrifice of the uninvolved part of the petrous pyramid.

ANATOMY OF THE PETROUS PORTION OF THE TEMPORAL BONE

Were otologists to make an organized effort for the purpose of confusing members of the medical profession at large on the subject of petrositis, they could not succeed better than they have to date with their disorganized application of nomenclature to the anatomic structures of the petrous portion of the temporal bone and to the disease of its various portions.

In this regard no two otologists speak of the same thing or employ the same terms, and few of them mean the same thing when they apply similar terms. An analysis of the current literature has convinced me that the confusion is due to the fact that the majority of authors derive their nomenclature from studies of anatomic textbook descriptions of the petrous pyramid, which unfortunately do not cover the present day needs of surgical intervention on the diseased pyramid.

Anatomists, with the exception of Guild,[2] have always described the petrous portion of the temporal bone as of one piece, truncated and pyramidal, with a base and an alleged apex and three surfaces. Today, when disease within different portions of the petrous pyramid is recognizable and specific surgical measures for the relief of localized pathologic processes are possible, the anatomic descriptions of the petrous

2. Guild, Stacy R.: Ann. Otol., Rhin. & Laryng. **44**:1011 (Dec.) 1935.

pyramid to be found in anatomic textbooks are inadequate for the requirements of surgical orientation. When talking of suppurations within the apex or the base of the petrous pyramid and recognizing the fact that they may appear circumscribed, as lesions which require specific and direct surgical treatment limited to surgical attack on this lesion only, it becomes obligatory to define clearly the surgical limits of the base and the apex of the petrous pyramid to determine which portion of the pyramid belongs to the apical section and which to the basal. It is therefore suggested that the petrous portion of the temporal bone be divided for the purpose of surgical orientation into two portions: (1) the basal labyrinthine portion and (2) the apical carotid portion.

The basal labyrinthine portion consists of: (1) the labyrinthine bony structure and (2) the structure made up of the perilabyrinthine cell groups. Each of these lends itself to independent surgical treatment. The basal labyrinthine portion presents for surgical inspection and intervention four surfaces:

1. The posterior fossa surface, which extends from the posterior semicircular canal up to and including the anteriormost limit of the opening of the internal auditory canal.
2. The superior middle fossa surface, extending from the eminentia arcuata of the superior semicircular canal up to the level of the beginning of the horizontal portion of the internal carotid canal.
3. The anterior, or tympanic, surface, extending from the external semicircular canal up to the vertical ascending portion of the internal carotid canal.
4. The inferior skull base surface, which forms the dome of the jugular bulb and extends up to the external opening of the internal carotid canal.

The geometric base of the basal labyrinthine portion of the petrous pyramid forms the mastoid aspect of the petrous portion of the temporal bone.

The apical carotid portion consists of: (1) the petrosal course of the internal carotid artery in its carotid canal and (2) the cellular bone structure lying partly superior and mostly posterior to the transverse course of the carotid canal. It presents for inspection and surgical intervention three surfaces:

1. The anterosuperior middle fossa surface, which extends from the beginning of the course of the transverse portion of the internal carotid artery through the carotid canal up to its exit from the petrous pyramid, through the internal orifice of the carotid canal in the very apex of the apical portion, to begin its ascent
2. The posterior fossa surface, extending from the anteriormost limit of the opening of the internal auditory meatus up to the very apex of the apical carotid portion of the petrous pyramid
3. The inferior skull base surface, which extends from the posterior limit of the external opening of the internal carotid canal up to the geometric apex of the apical carotid portion of the pyramid.

The geometric apex of the apical carotid portion of the petrous pyramid is received into the angular interval between the posterior border of the great wing of the sphenoid bone and the basilar part of the occipital bone.

This description of the petrous portion of the temporal bone and the nomenclature of the anatomic structures is here proposed for the purpose of clarification. It was arrived at from the surgical point of view and is therefore conducive to speedy surgical orientation within this compact field of delicate and vital anatomy.

TREATMENT OF PETROSITIS

The treatment varies with the part of the petrous pyramid pathologically involved.

Suppurative Labyrinthitis.—Suppurative petrositis involving and limited to the membranous labyrinth can be readily diagnosed. Suppurative labyrinthitis may either resolve and result in recovery with a permanently damaged internal ear or progress toward meningitis. When suppurative labyrinthitis involves the meninges, a labyrinthectomy performed before the bacteria invade the cerebrospinal fluid system will usually result in the recovery of the patient.

Suppurative Perilabyrinthitis.—Suppurative petrositis limited to the perilabyrinthine cell groups often presents a diagnostic problem which is difficult to solve. Though suppurative perilabyrinthitis when limited to the basal labyrinthine portion of the petrous pyramid may form one or more fistulous tracts and rupture through the superior or posterior surfaces, forming an epidural collection of pus in the middle or posterior cranial fossa or in both fossae, a definite diagnosis of such a condition is seldom made before an exploratory operation is performed. Although it is not easy to make an early definite diagnosis of suppuration of the perilabyrinthine cell groups, transient signs and symptoms may be recognized by a keen observer which are sufficient to warrant surgical exploration of the basal labyrinthine portion of the petrous pyramid. A most clarifying exposition of the diagnostic data in regard to suppurative perilabyrinthitis was contributed by Kopetzky [3] before the annual meeting of the American Otological Society, in Atlantic City, N. J., in April 1934.

When suppurative perilabyrinthitis is strongly suspected, the mastoid aspect of the base of the petrous pyramid should be severed from the mastoid process, and the basal labyrinthine portion of the petrous pyramid should be explored and its surfaces exposed and examined for fistulous tracts and epidural collections of pus. Fistulous tracts, when found, should be probed, and when they lead beyond the basal laby-

3. Kopetzky, S. J.: Ann. Otol., Rhin. & Laryng. **43**:718 (Sept.) 1934.

rinthine portion into the apical carotid portion, additional surgical intervention on the latter portion of the pyramid must be considered. This form of petrositis exists much more frequently than is suspected, and unless timely surgical intervention is undertaken it will often result in bacterial meningitis. One must deplore the temporizing attitude assumed by many otologists when they find themselves in the presence of signs and symptoms which though not distinctly pathognomonic or definitely localizing are nevertheless strongly suggestive and certainly warrant additional exploration of the temporal bone.

One or more groups of perilabyrinthine cells may become infected in suppurative perilabyrinthitis. The presence of a fistula in one of the surfaces of the basal labyrinthine portion of the petrous pyramid is no guarantee that there are not other additional fistulas on some other surfaces, and it is on this account that good surgical procedures attempted for the relief of suppurative perilabyrinthitis are usually incomplete unless all the surfaces of the basal labyrinthine portion are inspected and explored.

SUPPURATIVE APICITIS

Suppurative petrositis involving the apical carotid portion of the petrous pyramid may exist in the following forms:

1. Independent apicitis secondary to otitis media purulenta acuta without mastoiditis
2. Concurrent apicitis, occurring simultaneously with mastoiditis, secondary to otitis media purulenta acuta
3. Secondary apicitis resulting from suppurative perilabyrinthitis occurring some time after mastoidectomy

TREATMENT OF APICITIS

In independent apicitis the apical carotid portion of the pyramid is invaded directly through the tympanic cavity by an infection traveling anteriorly without traveling backward and therefore leaving the mastoid process uninvolved. This explains why, in many cases of otitis media purulenta acuta, with signs and symptoms of existing apicitis with or without paralysis of the abducens nerve, the mastoid process when surgically opened is found to be almost completely devoid of pathologic changes. In the vast majority of these cases the infection which invades the apical carotid portion of the petrous pyramid, though giving alarming symptoms and signs of apicitis, resolves immediately after the congestive stage of the infection without developing into the suppurative stage. In these cases of congestive apicitis recovery occurs after a simple mastoidectomy, but recovery occurs in many cases of congestive apicitis without surgical intervention on any part of the temporal bone. It is claimed that only about 20 per cent of these patients succumb to menin-

gitis when there is no surgical intervention on the pyramid. I am convinced that if in all the cases of independent apicitis repeated roentgenologic investigation were made, it would be found that the 80 per cent in which recovery takes place without surgical intervention on the petrous pyramid or after the often unnecessarily performed simple mastoidectomy, are those in which the lesion resolves before reaching the stage of suppuration. The 20 per cent in which death from meningitis occurs are those in which there is roentgenologic evidence of suppurative apicitis. To reduce the frequency of meningitis following this form of apicitis, with its admitted 20 per cent mortality rate, in addition to the surgical intervention on the mastoid process, it is necessary that surgical procedures be undertaken on the apical carotid portion of the pyramid. This becomes imperative in every instance of clinically diagnosed and roentgenologically proved suppurative apicitis.

With Kopetzky and Almour's [4] first report of the new, recognizable clinical picture, which described a new clinical entity and made possible the diagnosis of acute suppurative apicitis while the lesion is located in the pneumatic apical carotid portion of the petrous pyramid and which at the same time provided a safe surgical procedure for the drainage of infections therein, the otologic world suddenly became petrositis conscious. These observers stimulated a widespread discussion of this subject in the literature. Since a large amount of the discussion is still controversial, my views on the subject, based on a personal experience with all phases of petrositis, may perhaps throw some light on various aspects of the topic.

I find in reviewing my experience with the treatment of petrositis, that I passed through a transitional period, beginning with complete inactivity, a *laissez faire* attitude, masquerading as conservative otology. This was due to lack of knowledge of what to do in these cases other than a mastoidectomy. Then followed a second stage, with a sort of rationalizing self-defense conservatism, when I began to comprehend the pathologic picture of petrositis but did not know how to approach the lesion surgically. A third stage ensued when I became cognizant of the work of Eagleton,[5] Kopetzky and Almour,[4] Frenckner [5a] and

4. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid: Pathology, Symptomatology and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **39**:996 (Dec.) 1930; **40**:157 (March); 396 (June); 922 (Sept.) 1931.

5. Eagleton, Wells P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex: Report of Four Cases, with Recovery in Three, Arch. Otolaryng. **13**:386 (March) 1931.

5a. Frenckner, P.: Some Remarks on the Treatment of Apicitis With or Without Gradenigo's Syndrome, Acta oto-laryng. **17**:97, 1932.

Ramadier,[6] who described surgical procedures for drainage of suppurative apicitis. At this point I decided to employ the Kopetzky and Almour technic, because it appeared to me to be the simplest and safest of all those described, and it surgically drains any lesion of suppurative apicitis encountered in pneumatized bone.

From my personal evolutionary experience with apicitis and its sequelae the following conclusions have been reached as to the management and treatment of this condition:

Apicitis, like mastoiditis, does not always become suppurative; i. e., it may pass from the stage of congestion to that of resolution without ever becoming an actual suppuration. This type of apicitis does not require surgical intervention in the petrous pyramid any more than congestive mastoiditis needs surgical intervention within the mastoid process. When definite suppuration occurs within the apical carotid portion of the pyramid and is recognized clinically and roentgenologically, there is an imperative need for immediate surgical intervention within the apical carotid portion, provided the clinical evidence does not show that the process is in evident recession. I am fully aware that this view is not generally held by otologists. There are those who insist that suppurative apicitis does not require surgical therapy because they believe that in the vast majority of these cases recovery occurs by means of "natural" drainage through a fistula usually situated in the perieustachian tube cell region. There are those, on the other hand, who believe that surgical intervention is justified only when symptoms appear which are suggestive of bacterial invasion of the subarachnoid space.

I am profoundly convinced that in the treatment of acute suppurative apicitis there is to be no middle of the road course. Every case of definitely diagnosed acute suppurative apicitis should be directly attacked and surgically treated if there is to be a material reduction in the high incidence and mortality of suppurative meningitis consequent on purulent otitis media. I feel that, despite the fact that there are occasional cases of suppurative apicitis labeled "healed without surgical treatment," which nevertheless become chronic (as in many instances of suppurative mastoiditis), the so-called conservative method of abandoning suppurative apicitis to natural cure is poor surgical judgment or a surgical sin of omission, when the physician is aware that there are practical and, in many regards, ideal surgical procedures for the relief of this pathologic lesion. Nature may be a good doctor (as a famous scientist once remarked), but it is a mighty poor surgeon!

I am loath to believe that when Marvin F. Jones[7] stated that many cases of petrositis probably resolve following the simple operation of

6. Ramadier, J.: L'ostéite profonde du rocher, Oto-rhino-laryng. internat. **17**:816 (Nov.) 1933.

7. Jones, Marvin F.: Suppuration of the Petrosal Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1037 (Dec.) 1935.

myringotomy, and that "an adequate simple mastoidectomy must, in most cases, effect a resolution of the petrous involvement," he meant to indicate that definitely diagnosed suppurative apicitis can be cured by either myringotomy or simple mastoidectomy. In his broad application of the term petrositis Jones failed to specify what part of the petrous pyramid he referred to and also failed to designate the stage reached in the inflammatory process. It is necessary to emphasize the fact that direct surgical measures to the petrous pyramid should be considered only in the suppurative stage of an inflammatory process within the apical carotid portion of the petrous pyramid, and furthermore, one needs no reminder that apicitis in its first stage of inflammation, that is, in its stage of congestion, will often subside after myringotomy or mastoidectomy, because it may likewise subside without the employment of either of these procedures. Surgical measures to the petrous pyramid for congestive apicitis are not indicated any more than they would be for congestive mastoiditis. The issue is not "Is specific surgical operation advisable for petrositis?" but rather "Is specific surgical operation advisable for suppurative apicitis or suppurative perilabyrinthitis or both?" The answer is obvious.

Friesner and Druss,[8] in a paper read as part of a symposium before the American Otological Society, in Toronto, Canada, stated that "the presence of suppuration in the petrous pyramid is not, per se, an indication for immediate operation upon that structure." They likewise stated that "petrous pyramid suppurations are frequently found histologically in patients who die of otitic intracranial complications." It is obvious in a juxtaposition of these statements that their histologic findings do not lend much support to their initial contention, that "suppurative petrositis per se is not an indication for immediate operation upon the petrous pyramid." The fact that in many cases apicitis goes unrecognized and death occurs from bacterial suppurative meningitis is no argument against timely surgical intervention in instances of diagnosed suppurative apicitis but must rather be looked on as a definite indictment of diagnostic ability. The fact that persons with positively diagnosed suppurative apicitis do not always die when left alone is also not a scientific or logical reason for failure to intervene surgically. Does one await signs and symptoms of impending intracranial involvement before one intervenes surgically in acute suppurative mastoiditis, in which the chances for intracranial complications are much more remote? Why, then, should one hesitate to intervene surgically in definitely diagnosed suppurative apicitis without waiting for signs and

8. Friesner, I., and Druss, J. G.: Suppuration of the Petrosal Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1100 (Dec.) 1935.

symptoms of impending meningitis, when one knows that the risk of intracranial involvement in suppurative apicitis is much greater than in suppurative mastoiditis?

When surgical operation on the petrous pyramid is generally taught and practiced, better surgical judgment will supersede the present day halting, inadequate and often erroneous approach to the problem.

OPERATIVE PROCEDURE

When operative intervention is decided on, the most desirable surgical procedure is the next question. Various surgical methods have been described in the literature: Eagleton's unlocking of the petrous pyramid, Kopetzky and Almour's and Ramadier's drainage of the pneumatic petrous pyramid and Frenckner's perforation through the geometric base of the pyramid are among the best known.

These procedures, in general, while they permit an evacuation of accumulated pus, do not permit a complete exenteration of the lesion within the apical carotid portion of the petrous pyramid, nor do they afford a means of approach for drainage of an epidural abscess unless it spreads haphazardly into the operative field, particularly when an abscess is located in the posterior fossa, anterior or internal to the internal auditory meatus, which results from a rupture through the posterior wall of the apical carotid part of the pars petrosa. Furthermore, these procedures do not permit a full inspection of the entire interior of the apical carotid portion of the petrous pyramid.

While Ramadier's operation is essentially correct as far as the principle of surgical drainage of the contents of the apical carotid portion is concerned, it defeats its own purpose because drainage is afforded through an opening in the internal wall of the carotid canal, which opening becomes automatically obstructed when the carotid artery is allowed to fall back in situ.

The majority of otologists agree that when empyema of the apical carotid portion of the petrous pyramid is present (and in the absence of involvement of its confining walls), simple drainage through puncture, as performed by Kopetzky and Almour, is generally sufficient. This surgical principle presents itself likewise in empyema of the pleural cavity, but, nevertheless, there are many instances of pleural empyema which require extensive rib resection and extensive surgical opening to the pus cavity proper. In these cases healing does not follow simple thoracotomy. It must be understood that extrapetrosal surgical intervention, as suggested by Eagleton, is not practical because exposure of the apical carotid portion of the petrous pyramid through this route cannot be accomplished in the living subject without lacerating the dura. I cannot accept Eagleton's view that in every instance petrositis is osteomyelitis and should be treated similarly to infections of the long bones.

by perforation and drainage, because my own experience proves the contrary. Eagleton's view may be correct in a few instances, but I am convinced beyond doubt that his contention is not true of the majority of cases. I agree with Kopetzky's stand that suppurative apicitis occurs generally in the pneumatic type of the apical carotid portion of the petrous pyramid (except in infants) and that these pneumatized tips closely resemble in structure the pneumatized mastoid process (Guild). On the basis of this view, a surgical procedure providing for drainage of such an infection only is just as inadequate as Whiting contended it would be when used for the relief of suppurative mastoiditis.

These drainage procedures do not offer a complete solution to the problem of suppurative apicitis any more than Petit and Jasser solved the problem of suppurative mastoiditis when they drilled and probed the abscessed mastoid and established drainage without completely exenterating the pathologic processes. They lessened the factor which produced coalescence, and they undoubtedly saved lives by permitting a long-drawn-out procedure to relieve suppuration through the tract made by their trephine opening, but it hardly compares favorably with the quick results obtained by a complete modern mastoidectomy and its rapid healing. The same, I venture to predict, will occur when the results of apicectomy are compared with those of surgical drainage only.

While many cures of suppurative apicitis follow some of the aforementioned procedures, surgical treatment of the petrous apex will not be considered truly efficient and adequate until a procedure is employed which will afford inspection of the surfaces of the basal labyrinthine portion and likewise provide a means of opening the apical carotid portion by removing the entire anterosuperior wall of the apical part, thus enabling the surgeon to exenterate completely all cellular structure within the apical carotid portion and to expose and inspect the entire inner table, consisting of the posterior and inferior apical walls. By the employment of such a surgical procedure an epidural abscess in the posterior or middle fossa in the region of the apical carotid portion of the petrous pyramid may also be treated surgically. Necrosis and perforation of the inferior skull base surface of the apical carotid portion may likewise be treated by such a surgical procedure.

To follow the analogy with mastoidectomy—just as the operation had to replace the early antrotomy to obtain the desired results, so will apicectomy replace the present methods of apicotomy to obtain the maximum results with the lowest mortality in the treatment of acute suppurative apicitis.

Why should one expect that a diseased cellular apical carotid portion of the petrous pyramid will improve under incomplete surgical treatment when one does not expect a diseased mastoid process to do the same thing? The problem is not only to establish drainage, even though

in many cases it occurs to some extent through fistulous tracts, but additionally to remove successfully and easily all the pathologic tissue from within the apical carotid portion—in other words, to eradicate the lesion intrapetrosally.

The formation of abscesses within the apical carotid portion of the petrous pyramid may be multiple and each abscess walled off from the other by bony cellular structure or granulomas; thus drainage may be afforded to the nearest one, while the innermost and perhaps the most dangerous abscess is left untapped. The anatomic formation of the apical carotid portion may be partially sclerotic and partially pneumatic, and the drill or chisel may miss the necrotic pneumatic area. The petrous pyramid may be sclerotic, diploic or pneumatic or any combination of bone structure like the mastoid process. The type of cellular structure within the mastoid process is not always a clue to the type of structure in the apical carotid portion of the petrous pyramid. The generalization regarding the type of surgical procedure may find exception in the specific case with which the surgeon is confronted.

All otologists are aware with what frequency a mastoid process is opened and found normal looking, but the competent otologist always proceeds to explore the mastoid process completely and to search all the anatomic structures within it. Eventually he will find a walled-off abscess in the posterior perisinuous region, in the region of the emissary vein or in the zygomatic region.

The surgeon's results would be disastrous were he to desist immediately from other attempts after merely opening a mastoid process which failed to show pus. Incomplete surgical operation on the mastoid process is usually followed by disaster; why, therefore, should incomplete operation on the petrous pyramid be any safer? The contrary is undoubtedly true.

If suppurative apicitis is to be recognized as a clinical and pathologic entity which requires surgical intervention for relief, the surgical procedure instituted must be completely adequate to permit the eradication of the pathologic processes present. The surgical procedures so far described fail to provide such adequacy. They are all designed to establish additional drainage to the already existing drainage from a fistula, but none provides for the exenteration of the existing pathologic process under direct vision, i. e., for apicectomy. Apicectomy should be the present day operative procedure. The following case is cited to demonstrate that apicotomy is often inadequate:

D. S., a girl aged 7 years, had independent suppurative apicitis, the diagnosis of which was established clinically and corroborated by roentgenograms (Dr. Taylor). The mastoid process was opened and found not to be pathologically involved. After a mastoidotympanectomy was performed the apical carotid portion of the petrous pyramid was penetrated, and drainage was established by employing

the Kopetzky-Almour method. One-half teaspoonful (2 cc.) of pus escaped. Soon all the symptoms were relieved except the paralysis of the abducens nerve, which did not show improvement until the tenth postoperative day, when it suddenly disappeared. On the twelfth postoperative day she suddenly manifested definite signs and symptoms of acute suppurative meningitis. Examination of the spinal fluid at that time revealed a cell count of 1,680, 79 per cent polymorphonuclears, 21 per cent lymphocytes, a 4 plus reaction to tests for globulin and a trace of sugar; no organism was noted on direct smear but a long chain nonhemolytic streptococcus was found on culture. I decided to explore further and, if possible, to inspect and remove the lesion within the apical carotid portion of the petrous pyramid. It occurred to me that, since the internal carotid artery traverses the entire length of the apical portion of the petrous pyramid in the carotid canal, if I exposed the artery and followed its course by successive removal of the outer bony wall of the carotid canal and simultaneous extenteration of the cellular structure lying superior and posterior to its course I should be able to remove all the pathologic tissue and to expose the entire inner table of the apical carotid portion. By doing just that I found a large walled-off pus pocket in the apex of the apical carotid portion communicating with the posterior fossa through a perforation in the posterior wall, anterior to the internal auditory meatus.

This convinced me that puncture and drainage when employed as a surgical measure in suppurative apicitis is a nonsurgical procedure, because it does not permit a thorough search for and the complete exenteration of the pathologic process from within the apical carotid portion. After this experience I discarded the apicotomy and have since then employed the procedure for apicectomy exclusively in every case of suppurative apicitis, with the full knowledge that in many cases of suppurative apicitis recovery would follow apicotomy, just as in many cases mastoiditis would be cured after an antrotomy. It is the threat to life in which the occasional failure of apicotomy results that compelled me to employ the apicectomy in every case.

TECHNIC FOR APICECTOMY

The following description of the technic for apicectomy is quoted from my preliminary report.[1]

The surgical technic of an apicectomy may be divided into the following stages:

I. The performance of a mastoido-tympanectomy.

II. The exposure and inspection of the basal labyrinthine portion of the petrous pyramid.

III. The creation of a surgical intrapetrosal approach to the apical carotid portion of the petrous pyramid.

IV. The complete exenteration of the bony cellular structure of the apical carotid portion of the petrous pyramid accomplished through the exploration of the entire apical course of the internal carotid artery.

I. PERFORMANCE OF A MASTOIDO-TYMPANECTOMY (RADICAL MASTOIDECTOMY)

The performance of a mastoido-tympanectomy is essential to all surgery of the apical carotid portion whether apicotomy or apicectomy. It must be understood that extrapetrosal surgery is neither feasible nor practical because exposure of

the apical carotid portion of the petrous pyramid through this route cannot be accomplished in the living without lacerating the dura. The technic employed in a mastoido-tympanectomy is well known and needs no description. It will suffice to state that the end result of the mastoido-tympanectomy must consist of a complete exploration of the entire surgical anatomy within the mastoid process *and* the tympanic cavity. One must be able to demonstrate:

1. The entire inner bony table forming the floor of the middle fossa consisting of the tegmen tympani, tegmen antri and tegmen mastoidii.

2. The entire inner bony table covering the sigmoid sinus throughout its course within the mastoid process.

3. The inner bony table forming the anterior perisinus aspect of the posterior fossa (Trautmann's Triangle).

4. The inner bony table forming the posterior perisinus aspect of the posterior fossa.

5. The floor of the antrum and aditus.

6. The eminence of the external semi-circular canal.

7. The remaining lower limit of the tympanic portion of the posterior bony wall of the external auditory canal enclosing the descending portion of the facial nerve within the Fallopian canal, commonly known as the facial ridge.

8. The transverse tympanic portion of the facial nerve in its bony canal.

9. The oval window with stapes intact.

10. The promontory.

11. The semi-canal for the tensor tympani muscle.

12. The tympanic orifice of the Eustachian tube.

II. EXPOSURE AND INSPECTION OF BASAL LABYRINTHINE PORTION OF PETROUS PYRAMID

1. Exposure of the Geometric Base of the Petrous Pyramid: In a pneumatic mastoid process the mastoid aspect of the base of the petrous portion of the temporal bone, which fuses with the inner bony table of the mastoid process superiorly, posteriorly, and inferiorly and the anterior and outermost limit of which is marked by the eminence of the external semi-circular canal, most often is composed of perilabyrinthine groups of cells. These perilabyrinthine cells in the regions inferior, superior, and posterior to the mastoid aspect of the base of the petrous pyramid plus the cells in the solid angle are removed until the mastoid aspect of the petrous base with the external, posterior, and superior semi-circular canals are sharply defined.

2. The Severance of the Petrosal Base from the Mastoid Process: This is accomplished by removing the inner bony table of the mastoid process covering the sigmoid sinus throughout its course; also, removing the entire inner bony table which forms the roof of the mastoido-tympanic cavity and exposing the dura of the middle fossa which covers the temporal lobe. The inner bony table which occupies a position supero-anterior to the sinus in Trautmann's Triangle and infero-anterior to the sigmoid sinus between it and the facial ridge up to the base of the petrous is also removed thus exposing the dura of the posterior fossa.

3. Inspection of the Basal Labyrinthine Portion of the Pars Petrosa: By gently separating the dura in the posterior fossa from the posterior surface of the petrous base, the basal labyrinthine portion lying internal and anterior to the posterior semi-circular canal is examined for pathologic changes. The dura of

the middle fossa is gently elevated from the superior surface of the basal labyrinthine portion, and a search for pathologic changes is made internal and anterior to the eminentia arcuata. The anterior or tympanic wall of the basal labyrinthine portion of the petrous pyramid is searched for a supra- or infra-cochlear fistula.

III. CREATION OF AN APPROACH TO THE APICAL CAROTID PORTION OF THE PETROUS PYRAMID

In order to create a practical adequate surgical field of approach to the apical carotid portion of the petrous pyramid, one that will be spacious enough to permit free instrumentation and surgical manipulation under direct vision aided by proper illumination, one must do the following:

1. Supplement the already existing soft part incision in order to bring into full view the *anterior* bony wall of the external auditory canal.

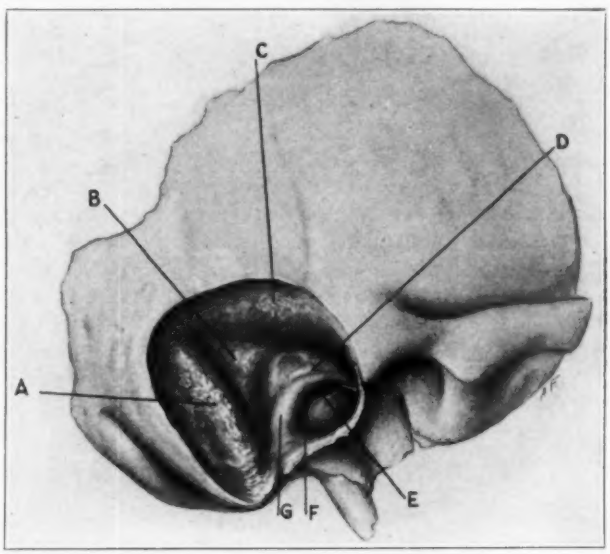

Fig. 1.—Mastoidotympanectomy (radical mastoidectomy). *A* indicates the tegmen of the lateral sinus; *B*, the tegmen of the posterior fossa; *C*, the tegmen of the middle fossa; *D*, the external semicircular canal; *E*, the oval window, with the stapes intact; *F*, the round window, and *G*, the facial ridge with the descending portion of the facial nerve.

a. An initial incision is made in the uppermost part of the anterior wall of the external auditory canal, where its cartilaginous portion is attached to its bony part and is then carried from above downward and from within outward, along the entire line of attachment until it emerges from the external auditory canal at the junction of the inferior and anterior canal walls. Returning to the starting point, the incision is carried through skin and periosteum down to the squama, upward and outward into the triangular non-cartilaginous space between the tragus and helix, hugging the anterior wall of the helix for a distance of about three-quarters of an inch. Retractors are then introduced into this latter portion of the incision and the auricle is retracted backward over the mastoid

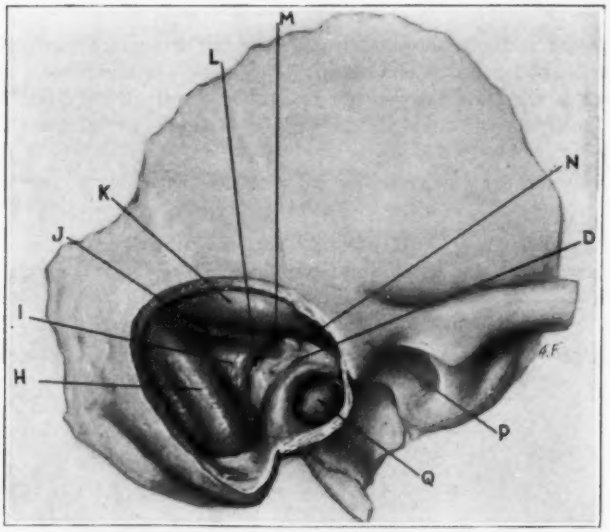

Fig. 2.—Exposure of the basal labyrinthine portion of the petrous pyramid, accomplished by (1) the exenteration of the perilabyrinthine cells covering the mastoid aspect of the basal portion of the petrous pyramid and (2) the removal of the entire inner table of the mastoid process, thus severing the base of the pyramid from the mastoid process. *H* indicates the wall of the lateral sinus; *I*, the dura of the posterior fossa; *J*, the superior petrosal sinus; *K*, the dura of the middle fossa; *L*, the posterior semicircular canal; *M*, the solid angle; *N*, the superior semicircular canal; *D*, the external semicircular canal; *P*, the posterior root of the zygoma, and *Q*, the promontory.

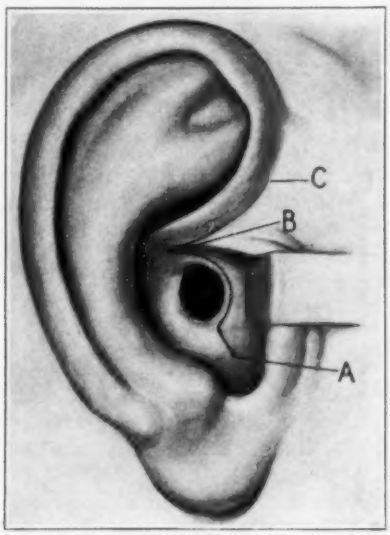

Fig. 3.—Supplementary incision for apicectomy. *AB* indicates the incision in the anterior wall of the external auditory canal and *BC* the incision over the squamous portion of the temporal bone.

region. Such a posterior displacement of the auricle permits of a surgical approach to the apical carotid portion of the petrous pyramid, and reduces to a minimum the distance existing between the apical carotid portion and the outermost edge of the wound.

b. The technic to be described can also be carried out through the previous postauricular incision by extending this incision forward and upward along the squama. The anterior membrano-cartilaginous canal wall is then separated from the bony canal and drawn forward with the auricle. The disadvantage here is that approximately one-half inch of soft tissue, plus the tubular membrano-cartilaginous canal is added to the distance normally existing between the outer surface of the skin and the apical carotid portion of the petrous pyramid.

2. Bring the Tympanic Orifice of the Eustachian Tube into Full View: The entire posterior root of the zygoma, which enters into the formation of

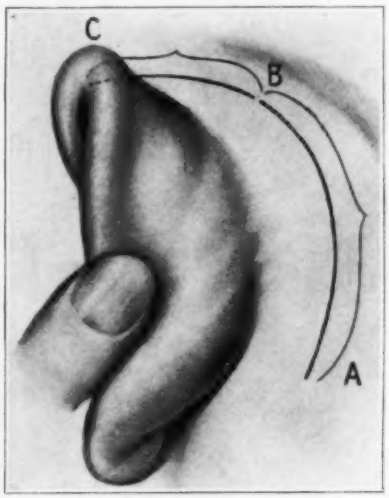

Fig. 4.—Supplementary incision for apicectomy. *AB* indicates the already existing postauricular incision, and *BC* the extension of the postauricular incision over the squamous portion of the temporal bone.

the outer part of the supero-anterior wall of the external auditory canal is removed with an electrically driven round burr, or with hammer and chisel. The remainder of the tegmen tympani is removed by lifting it with a curette until the mouth of the Eustachian tube and the semi-canal for the tensor tympani muscle are fully exposed.

The orifice of the semi-canal for the tensor tympani muscle, and the tympanic orifice of the Eustachian tube are both situated at the upper part of the anterior wall of the tympanic cavity and are separated from each other by a thin horizontal plate of bone, known as the *septum canalis musculotubaris*. The semi-canal for the tensor tympani lies beneath the tegmen tympani and superior to the Eustachian tube orifice.

3. Remove the Lower Border of the Squama: Approximately one-half inch area of the lower border of the squama is removed, beginning anteriorly above the

posterior end of the zygoma proper and ending with the posterior limit of the lower border of the squama. This step in the procedure permits a certain amount of retraction of the temporal lobe which affords added space for instrumentation during the surgical intervention within the apical carotid portion of the pars petrosa.

4. Remove the Peri-Eustachian Tube Cells: The peri-Eustachian tube cells are exenterated, and the mouth of the Eustachian tube is widened by the destruction of the septum canalis musculotubaris.

5. Destroy the semi-canal for the Tensor Tympani muscle and remove the muscle.

6. Expose the Tympanic, Ascending Vertical Portion of the Internal Carotid Canal: With a subperiosteal elevator inserted anterior to the outer edge of the bony anterior wall of the external auditory canal, and hugging its anterior, or

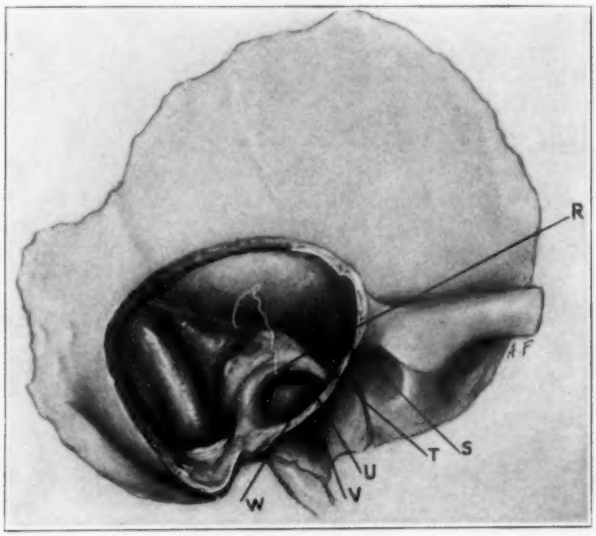

Fig. 5.—Creation of an approach to the apical carotid portion of the petrous pyramid. In step 1 the tympanic orifice of the eustachian tube is brought into full view by the removal of the entire posterior root of the zygoma, the removal of the perieustachian tube cells and the destruction of the semicanal for the tensor tympani muscle. In step 2, beginning with the posterior end of the zygoma proper, a ½ inch (1.25 cm.) area of the lower border of the squama is removed. *R* indicates the transverse tympanic portion of the facial canal; *S* the anterior articular part of the mandibular fossa; *T*, the petrotympanic fissure; *U*, the tympanic orifice of the eustachian tube; *V*, the posterior nonarticular part of the mandibular fossa, and *W*, the semicanal for the tensor tympani muscle.

mandibular surface, the soft tissues lying adjacent, internal, and anterior to it are gradually and progressively separated and displaced anteriorly toward the mandible. As one carries out the separation, one simultaneously and gradually removes piecemeal (with the aid of a narrow curved Rongeur and the electrically

driven round burr) the entire tympanic bony part of the anterior wall of the external auditory canal, plus part of the post-glenoid process (which enters into its formation above and anteriorly), the entire anterior and inferior parts of the annulus tympanicus, and as much of the tympanic bony wall of the posterior non-articular part of the mandibular fossa as is required to reach the petrotympanic fissure. With the insertion now of a narrow, deep-reaching blunt retractor and the displacement of the soft tissues anteriorly toward the mandible, approximately one-half inch of additional surgical space is gained, thus bringing the apical carotid portion still closer to the operator. Owing to the anatomic fact that the posterior half of the mandibular fossa is not concerned with articulation and that this posterior part of the glenoid fossa is filled with soft tissue and

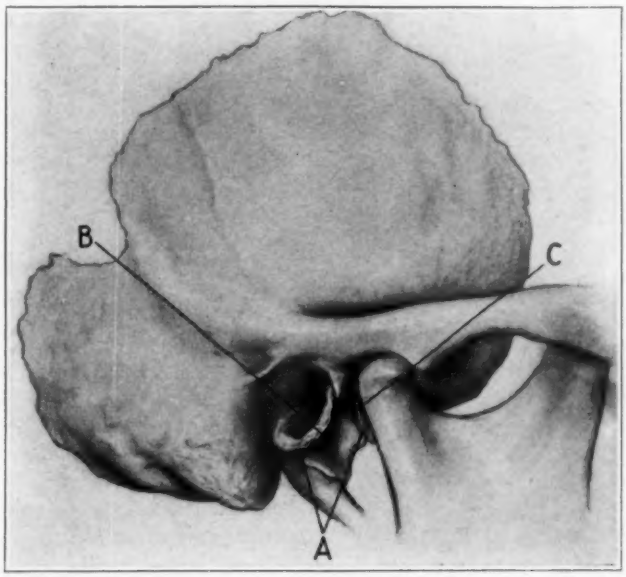

Fig. 6.—Existing surgical space between the nonarticular posterior part of the mandibular fossa and the mandible. *A* indicates the space between the mandible and the tympanic bony anterior canal wall resulting from the anatomic displacement backward of the nonarticular part of the mandibular fossa; *B*, the nonarticular part of the mandibular fossa and *C*, the petrotympanic fissure.

sometimes by a portion of the parotid gland, and, that these tissues are easily compressible, this step in the surgical technic of apicectomy is accomplished with ease, giving the surgeon additional freedom of action.

At the completion of this step in the technic we have exposed to direct view and within reach for instrumentation, and also for further surgical intervention, the entire tympanic ascending vertical portion of the internal carotid artery lying in its bony canal, anterior and internal to the promontory. The carotid canal is the most important anatomic structure which has to be exposed in the performance of an intrapetrosal apicectomy.

IV. THE COMPLETE EXENTERATION OF THE BONY CELLULAR STRUCTURE OF THE APICAL CAROTID PORTION OF THE PETROUS PYRAMID ACCOMPLISHED THROUGH THE EXPLORATION OF THE ENTIRE APICAL COURSE OF THE INTERNAL CAROTID ARTERY

Before describing this step it is necessary to call attention to the anatomic position and the course of the internal carotid artery in its canal, in its relation to the petrous portion of the temporal bone. The internal carotid artery in its course through the apical carotid portion of the petrous pyramid is enclosed in a bony canal. This canal has an external and an internal orifice. The external orifice, which is the beginning of the carotid canal through which the internal carotid artery enters the petrous portion of the temporal bone, is located on the inferior surface of the apical carotid portion of the petrous bone, while the internal or anterior orifice of the carotid canal is in the very apex of the apical

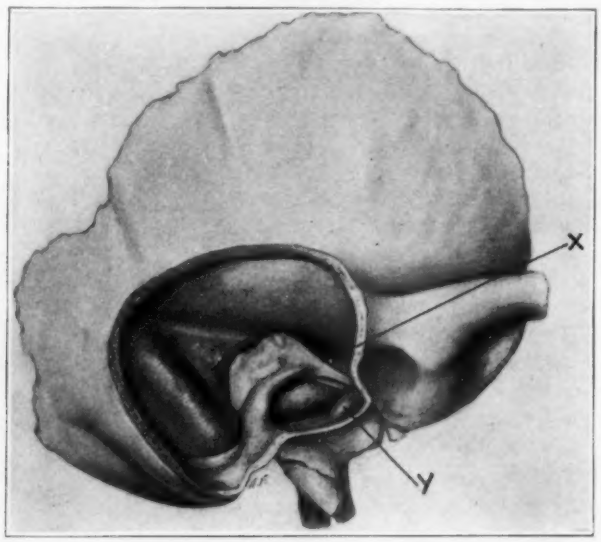

Fig. 7.—Creation of an approach to the apical carotid portion of the petrous pyramid. In step 3 the tympanic ascending vertical portion of the internal carotid canal is exposed by removing the tympanic bony wall of the posterior nonarticular part of the mandibular fossa, which forms the anterior wall of the external auditory canal down to the petrotympanic fissure. *X* indicates the superior bony middle fossa surface of the basal labyrinthine portion of the petrous pyramid and *Y*, the vertical tympanic portion of the internal carotid canal.

carotid portion. The petrous geometric apex is received into the angular interval between the posterior border of the great wing of the sphenoid and the basilar part of the occipital.

The carotid canal runs along the entire antero-superior wall of the apical carotid portion of the petrous pyramid. At first it ascends vertically and then bends a short distance anteriorly and internal to the promontory; this part of the carotid canal is known as the ascending vertical portion. At the termination of its vertical course the carotid canal curves forward and medialward, and runs along the entire length of the antero-superior wall of the apical carotid portion

of the petrous pyramid. When the internal carotid artery leaves the carotid canal through its internal orifice to enter the cavity of the skull, between the lingula and the petrosal process of the sphenoid, it again ascends.

This course of the internal carotid artery through the apical carotid portion is of great importance and assistance in the performance of a complete apicectomy. *A complete exposure of the internal carotid artery and its employment as a guide is the only way to reach and exenterate successfully every part of the apical carotid portion of the petrous pyramid, and, the best means to avoid injury to the internal carotid artery.* (Just as the sigmoid sinus in its course through the mastoid portion of the temporal bone acts as a guide to complete exenteration of the mastoid process, so must the internal carotid artery in its course through the apical portion of the petrous pyramid act as a guide to complete extenteration of the apical carotid portion of the petrous pyramid. No apicectomy is complete or can be completed without the exploration of the entire apical course of the internal carotid artery, just as no mastoidectomy is complete or can be completed without the exploration of the entire course of the sigmoid sinus in the mastoid.) In my experience the peri-carotid plexus of veins, described by anatomists, was never encountered during the process of removal of the external bony wall of the internal carotid canal. The exposure of the internal carotid artery was not attended by bleeding (in the cases upon which I operated).

We remove the outer bony wall of the carotid canal with a sharp curette, beginning with the ascending tympanic end, and expose the artery throughout its vertical course up to its bend. The bony plate forming the outer or external wall of the carotid canal in this region is very thin and very often dehiscent in parts. The bony plate is gently and gradually lifted, with the cup of the curette directed away from the arterial wall, until the canal curves forward and medialward to begin its horizontal course along the antero-superior wall of the apical carotid portion of the petrous pyramid. At this point begins the surgical antero-superior middle fossa surface of the apical carotid portion.

The antero-superior wall of the apical carotid portion above the horizontal course of the carotid canal from its bend forward to the very tip, slopes upward and backward forming a triangulation with the posterior wall, thus creating a tent-like space, with the inferior surface of the apical carotid portion as its base. It is in this triangular tent-like space that the cellular structure is to be found lying partly superior to and mostly posterior to the horizontal portion of the carotid canal. The cell exenteration of this space within the apical carotid portion of the petrous pyramid is begun at a point where the tympanic ascending portion of the carotid artery bends and begins its horizontal apical course forward and medialward. With a small, sharp curette, with the edge of the cup directed away from the carotid artery, we slowly continue the exposure of the carotid and continue the removal of the outer carotid canal wall one mm. spicule at a time, and follow each mm. of bony canal wall with one mm. of cell removal, by curetting superoposteriorly to the upper edge of the inner wall of the carotid canal. *Taking advantage of the fact that the internal carotid artery in its never varying course through the petrous portion of the temporal bone marks the beginning and end of the apical carotid portion of the pars petrosa, we perform a complete apicectomy by exposing and following the artery.* We follow its course through the apical portion by progressively lifting small portions of the outer bony carotid canal wall and alternately exenterating the cellular structure directly superior and posterior to the course of the artery until the entire apical portion of the carotid artery is exposed up to its upward bend where it makes its exit from the very apex of the apical carotid portion. (All the apical cellular structure with all its

contained lesions is thus exenterated. In order to get a clear view of the floor of the apex it is necessary to remove some bony structure lying between the cochlea and the vertical portion of the carotid. This bony area is most often eburnized and gives the impression, or appearance, of the cochlear anterior wall fusing with the posterior wall of the vertical carotid canal but actually this is not so. There is always an area of at least 2 mm. of bone separating or fusing the cochlea with the wall of the carotid canal. This area can be removed with the curet, but often the drill or chisel is needed. This area of bone is removed until the floor of the apex is exposed on a level with the jugular dome.)

In the process of curetting the apical cellular area we progressively remove the cortex of the entire antero-superior middle fossa wall of the apical carotid portion away from the dura covering the temporal lobe, with a curette and a fine-pointed Rongeur. By this process of progressively lifting and removing minute portions of cortex away from the dura we avoid injury to the dura. The temporal lobe is then gradually retracted, thus enlarging the field and facilitating surgical instrumentation. The inner table of the apical carotid portion (which is formed by the bony posterior fossa wall of the apical carotid portion lying internal to the internal auditory meatus) is now inspected for the presence of a fistula. This wall, if necrotic, is curetted gently and lifted away piecemeal from the posterior fossa dura and if necessary entirely removed.

The inner bony table of the floor of the apical carotid portion, which is formed by the base of the skull, is also inspected, and if diseased surgically treated.

By deliberately opening the carotid canal and exposing the artery throughout its petrous course an existing pericarotid collection of pus is readily drained. In an apicectomy for acute bacterial meningitis (by deliberately removing the posterior bony wall of the apical carotid portion and thus exposing the dura of the posterior fossa) a direct approach for surgical drainage of the cisterna pontis and the cisterna interpeduncularis is provided.

The technic described was carried out in four cases of definitely preoperatively diagnosed suppurative apicitis. In two of these cases suppurative apicitis was already complicated by bacterial suppurative meningitis when the apicectomy was first instituted by me. The complete apicectomy was also performed in five cases of definitely diagnosed bacterial suppurative meningitis.

In any case of otitic bacterial suppurative meningitis in which the immediate focus of infection had not been located previous to its onset apicectomy was performed with a twofold purpose: (1) to try to locate a possibly existing focus of infection within the petrous pyramid or in the intracranial structures surrounding it which has escaped diagnosis and detection previous to the onset of meningitis and (2) to remove the posterior wall of the apical carotid portion of the petrous pyramid, anterior and internal to the internal auditory meatus, thus exposing the dura in the posterior fossa and permitting the incision of this dura to drain the cisterna pontis infratentorially or the cisterna interpeduncularis supratentorially.

REPORT OF CASES

CASE 1.—D. S., a girl aged 7 years, was admitted to the hospital on Aug. 2, 1934. She gave a history of illness since April 29, beginning with lobar pneumonia on the right side, followed by lobar pneumonia on the left side five days later. Empyema developed on the left side, and rib resection was performed on the twenty-second day of May. Otitis media purulenta acuta developed on the left side ten days later; three weeks previous to her admission, she began to complain of severe headaches and pain back of the left eye and paralysis of the abducens

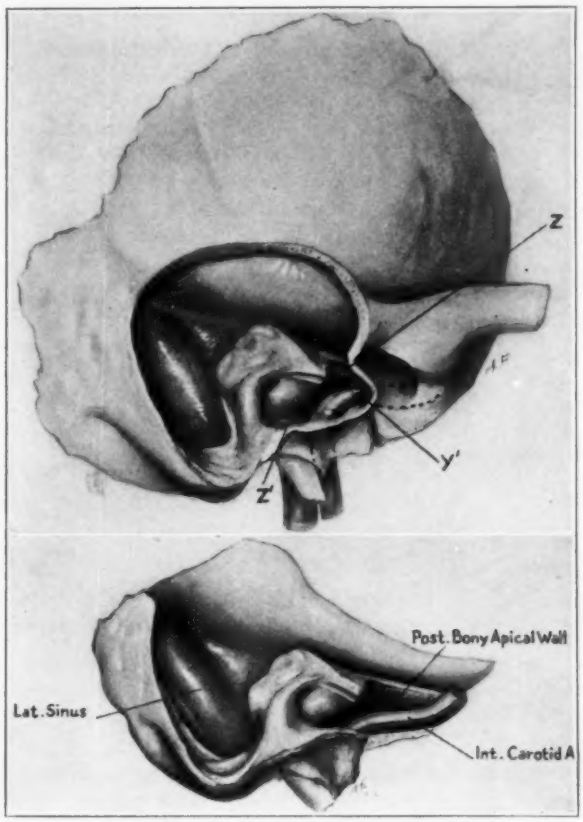

Fig. 8.—The upper figure shows the complete exenteration of the apical carotid portion of the petrous pyramid accomplished by the complete exploration of the entire apical course of the internal carotid artery. *Y'* indicates the transverse apical portion of the internal carotid artery; *Z*, the bony wall of the posterior fossa of the apical carotid portion of the petrous pyramid, and *Z'* the jugular dome.

The lateral sinus and the internal carotid artery as shown in the lower part of the figure are the guides to complete surgical intervention on the temporal bone. No mastoidectomy is complete without a complete exploration of the entire course of the transverse sinus in the mastoid. No apicectomy is complete without a complete exploration of the entire course of the internal carotid artery in the petrous pyramid.

nerve suddenly developed. She vomited frequently, and the aural discharge was scant. On the day of admission to the hospital the paralysis of the abducens nerve was marked, the pain in back of the eye severe and the headache marked. The discharge from the middle ear was purulent and scant. No sagging and no tenderness of the mastoid were observed. The blood count on admission showed: erythrocytes 3,900,000, leukocytes 21,000, with polymorphonuclears 80 per cent and small lymphocytes 20 per cent, and hemoglobin content 60 per cent. Urinalysis showed definite evidence of existing pyelitis. The temperature on admission was 101.6 F.; the spinal fluid on admission showed a normal pressure, 1 cell, a 3 plus reaction to tests for globulin and a trace of sugar; no bacteria were noted on smear or culture.

A roentgenogram of the chest revealed a hydropneumothorax on the left side, with partial collapse of the lung. There was a small effusion in the base of the left lung, with a plastic exudate on the parietal pleura. The upper lobe and the basal portion of the lower lobe of the left lung showed diminished aeration. This was due to an area of incomplete consolidation, with areas of atelectasis. There was slight shifting of the mediastinum to the left. Because of

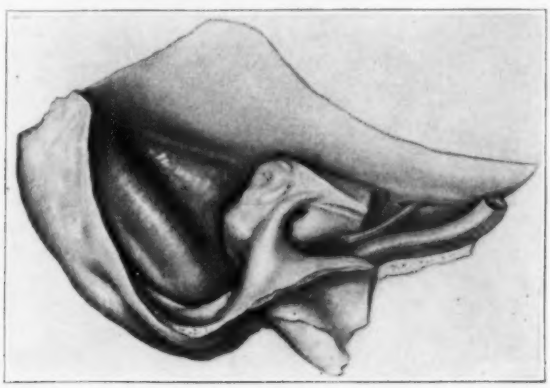

Fig. 9.—Drainage of the cisterna pontis.

the duration of the pulmonary lesion, an investigation was suggested for a nonopaque foreign body in the left lung. There was no evidence of involvement of the right lung.

Roentgenograms of the mastoids and the petrous pyramids (Dr. Taylor) revealed no definite signs of suppurative mastoiditis; a well defined abscess cavity in the apical carotid portion of the left petrous pyramid was readily demonstrable. I performed a mastoidotympanectomy on August 2 and found free pus in the antrum. The mastoid cell structure showed no evidence of suppuration. The tympanic cavity was filled with polypi and granulations, and the perieustachian tube cells were necrotic. The child showed no improvement whatever. On August 11 an apicotomy was performed by the Almour method, and about 1 teaspoonful of thick pus escaped through the fistula thus created. A drain was inserted. Immediately after the puncture and drainage of the apical portion of the petrous pyramid the retro-orbital pain and the general headache disappeared, but the paralysis of the abducens nerve showed no tendency to subside. The child, however, showed general improvement up to August 21, when the paralysis of the abducens nerve, which had persisted all the time, suddenly disappeared.

Two days later she suddenly manifested definite symptoms and signs of acute suppurative meningitis. Examination of the spinal fluid on August 23 revealed a cell count of 1,680, with polymorphonuclears 79 per cent and lymphocytes 21 per cent, a 4 plus reaction to tests for globulin, a trace of sugar, no organism on direct smear and a long chain nonhemolytic streptococcus on culture.

With the aforementioned findings of bacterial suppurative meningitis, I decided to explore the apical carotid portion of the petrous pyramid. On August 24 I performed a complete apicectomy and found a walled-off abscess in the very apex of the apical carotid portion of the petrosa, which was being prevented from draining through the Almour fistula by a large granuloma. The posterior wall of the apical carotid portion was necrotic and perforated. An epidural collection of pus in the posterior fossa was encountered and drained.

In my anxiety to save this child I was not satisfied to leave her alone when the existing focus of infection was definitely located and removed and decided to institute intracarotid therapy at once. Pregl's iodine solution and acriflavine base were injected alternately every six hours into the exposed petrosal course of the internal carotid artery.

On September 2 after an intracarotid injection of Pregl's iodine solution, the child suddenly died from a cerebral embolus.

Comment.—Having explored and exenterated the pathologic process within the apical carotid portion of the petrosa, thus removing the existing cause of the bacterial meningitis, I should have been satisfied to treat this child with supportive measures, such as daily small blood transfusions. Instead, I overtreated her by injections of Pregl's iodine solution alternating with a solution of acriflavine base every six hours into the exposed petrous portion of the internal carotid artery in addition to the daily blood transfusions. The puncture of the wall of the internal carotid artery four times daily undoubtedly resulted in thrombosis with the ultimate cerebral embolism, which was the cause of sudden death at a time when the spinal fluid showed definite signs of the bacterial suppurative meningitis abating.

The history, clinical course, pathologic findings and treatment in this case prove several points: 1. Suppurative apicitis can exist independently, without suppurative mastoiditis. 2. Mastoidectomy does not cure suppurative apicitis. 3. In some cases apicitis cannot be cured even by apicectomy.

Both the clinical course and the ultimate pathologic findings in this case give one the right to assume that if a complete apicectomy had been performed immediately when the diagnosis of suppurative apicitis was made, and the apical carotid portion of the petrous pyramid thoroughly explored and exenterated under direct vision instead of punctured blindly and drained, acute bacterial suppurative meningitis perhaps would never have resulted.

Case 2.—A. P., a man aged 50, was admitted to the hospital on April 17, 1935 with a history of otitis media purulenta chronica on the right of twenty-five years' duration. Three weeks previous to admission he had an acute exacerba-

tion of the otitis media purulenta chronica, which was ushered in with a profuse purulent discharge from the middle ear, severe headache and dizziness. Two weeks previous to admission he began to complain of pain back of the right eye, which increased in severity every day. The temperature ranged during the three weeks from 99 to 101.5 F. On the day of admission the blood count showed 5,110,000 red blood cells and 17,900 leukocytes, with 60 per cent polymorphonuclear cells, 29 per cent large lymphocytes, 6 per cent large mononuclear cells, 2 per cent transitional cells, 2 per cent eosinophils and 1 per cent basophils; the hemoglobin content was 84 per cent. Examination of the ear revealed absence of the tympanic membrane, a profuse purulent pulsating discharge from the middle ear and no tenderness over the mastoid.

Roentgen examination of the right temporal bone, done by Dr. Henry K. Taylor, revealed a pneumatic and partly sclerotic mastoid process, with evidence of chronicity; the right petrous pyramid revealed definite evidence of suppuration and necrosis of the bone within the apical carotid portion of the petrous pyramid, which was pneumatic. A diagnosis was made of suppurative apicitis, following an acute exacerbation of otitis media purulenta chronica on the right side. A complete mastoidotympanoapicectomy was performed.

Operation and Course.—No pathologic involvement of the mastoid process was noted. The mastoid antrum was filled with pus and granulations. The cells, which were partly pneumatic and partly sclerotic, showed no evidence of suppuration. A fistula in the region between the bend of the vertical tympanic portion of the internal carotid canal and the cochlea was found. A probe introduced into this fistulous tract led for a distance of about ½ inch (1.25 cm.) into the apical carotid portion of the petrous pyramid. Although pus was exuding from the fistulous tract, it did not flow freely. The apical carotid portion of the petrous pyramid was widely opened by completely exploring and following the internal carotid artery, and the entire cellular region superior and posterior to the artery was completely exenterated. A large abscess cavity was found in the extreme apex of the apical carotid portion. A perforation was found in the posterior bony wall of the apical carotid portion anterior to the internal auditory meatus, with an epidural collection of pus in the posterior fossa. The exenteration was completed; a gauze drain was inserted and the apical carotid portion was packed lightly. Immediately after this surgical intervention, the headache, dizziness and retro-orbital pain disappeared. On the third day after the operation erysipelas suddenly developed, which complicated his convalescence. This lasted until April 30, when his fever receded and after which he made an uneventful recovery. He was discharged from the hospital on May 8, 1935. His mastoidotympanoapical wound healed within eight weeks after the operation. A culture of the pus from the apical abscess revealed Streptococcus haemolyticus.

Comment.—This case is one in which chronic apicitis may have been in existence for many years, which was the cause of the persisting otitis media purulenta chronica for the last twenty-five years. An acute exacerbation of this otitis media purulenta chronica gave rise to an acute reinfection of the cellular structure within the apical carotid portion of the petrous pyramid, which resulted in suppuration. The mastoid process, which had undergone partial sclerosis, resisted the infection and remained uninvolved by this new invasion.

CASE 3.—S. R., a boy 5 years of age, was admitted to the hospital on June 13, 1935, with a diagnosis of acute bacterial suppurative meningitis of otitic origin.

History.—The patient had had scarlet fever three months before examination and otitis media purulenta acuta on the right side, two weeks after the onset of scarlet fever. A Schwartze mastoidectomy on the right was performed on May 15, 1935. On the eleventh postoperative day he began to have fever, the temperature, ranging between 101 and 103 F. He complained of headache and pain in the right eye and photophobia. Drainage from the mastoid wound and from the middle ear remained profuse and purulent. On June 12 definite signs and symptoms of acute suppurative meningitis manifested themselves.

Examination of the spinal fluid on June 13, the day of admission, revealed a cell count of 1,860, Str. haemolyticus on both smear and culture and no sugar. Roentgenographic examination of the petrous pyramids was done by Dr. S. E. Sinberg, who reported as follows: "Both petrous pyramids show moderate pneumatization. There is no evidence of any breaking down or productive changes in either bone. Several intact cells can be seen in the right mastoid region, which is generally cloudy." On June 14 I revised the mastoidectomy wound and performed a mastoidotympanectomy. Evidence of incomplete exenteration of the mastoid process was found, in the form of a completely intact necrotic zygomatic cell region, with many necrotic cells in Trautmann's triangle, in the tip and in the posterior perisinuous cell region. The tympanic cavity was full of pus and granulations. An examination of the spinal fluid on June 14 showed a cell count of 2,143; Str. haemolyticus was noted on smear and culture; sugar was absent. The child was getting progressively worse. On June 15 I performed a complete apicectomy with the object of searching for a possible focus of infection in the petrous pyramid, which the clinical picture strongly suggested but, which could not be substantiated by roentgenograms. Another reason for performing the complete apicectomy was my desire to drain surgically the cisterna interpeduncularis and the cisterna pontis. An exploration of the internal carotid artery in the carotid canal revealed a pulsating pericarotid collection of pus within the carotid canal. Part of the external carotid canal wall was missing, and the dura of the middle fossa resting on the anterosuperior wall of the apical carotid portion of the petrous pyramid was inflamed and thickened. The cellular area lying superior and posterior to the transverse course of the internal carotid artery within the apical carotid portion of the petrous pyramid was soft and necrotic but not abscessed. After complete exenteration of the cells, the posterior bony wall of the apical carotid portion, which occupies a position anterior and internal to the internal auditory meatus, was removed, and the dura of the posterior fossa was exposed. The dura was incised infratentorially, and the cisterna pontis was thus entered. A large amount of purulent spinal fluid escaped. A piece of fine rubber tubing was inserted into the cisterna pontis. The wound in the apical portion was packed lightly with narrow strips of gauze. One strip of gauze was tied to the tubing to safeguard its position. One hundred cubic centimeters of blood was given intravenously. An intracarotid injection of Pregl's iodine solution was given into the lumen of the exposed apical portion of the internal carotid artery.

On June 16 the child showed marked improvement. Orbital pain disappeared, and the rigidity of the neck and the Kernig sign were less marked. The spinal fluid on June 16 showed 823 cells, no organism on smear and Str. haemolyticus on culture. A trace of sugar was noted. Daily injections of Pregl's iodine

solution alternating with antistreptococcus serum were given into the internal carotid artery. The antistreptococcus serum was given because of the history of scarlet fever. Daily transfusions of 100 cc. of blood were given. The child continued to improve.

The spinal fluid was examined daily thereafter. The results were as follows:

Date	Cell Count	Organism on Smear	Organism Cultured	Sugar, Mg. per 100 cc.
June 17	560	None	Str. haemolyticus	20
June 18	542	None	Str. haemolyticus	28
June 19	450	None	Str. haemolyticus	30
June 20	289	None	None	38
June 21	187	None	None	45
June 24	90	None	None	55

On June 25, after the routine intracarotid injection of Pregl's iodine solution, cerebral embolism occurred. The patient died.

Comment.—This case was very instructive because it revealed the following points:

1. Suppurative apicitis may exist in the form of a pericarotid collection of pus within the carotid canal, which travels and extends along the internal carotid artery. Because the cellular structure lying superior and posterior to the internal carotid canal did not undergo suppuration and abscess formation, the roentgenogram was of no assistance in the diagnosis in this case.

2. When a pericarotid collection of pus exists without accompanying empyema of the cellular structure of the apical carotid portion of the petrous pyramid, the roentgenographic examination is of no assistance in the diagnosis of such a condition.

3. The importance and significance that should have been attached to the clinical picture which existed in this case for two weeks prior to the onset of meningitis, the lack of roentgen evidence of any existing suppuration within the apical carotid portion of the petrous pyramid and the ultimate discovery of a pericarotid abscess on the operating table, all tend to prove that there is still much research to be done on the subject of suppurative apicitis.

4. The progressive recovery that this patient was making, as evidenced by the continuous improvement of the cerebrospinal fluid and of the clinical picture, strengthened my belief that suppurative bacterial meningitis of otitic origin when observed early may be cured by surgical drainage of the cisterna interpeduncularis (supratentorial) or the cisterna pontis (infratentorial) or both, provided one succeeds in locating and eradicating the focus of infection in the temporal bone which acts as the inciting cause of the meningitis.

5. Intracarotid injections of Pregl's iodine solution and acriflavine base solution are dangerous. I have employed intracarotid therapy in

four cases of bacterial suppurative meningitis, and no beneficial results therefrom were observed at any time. Twice death resulted from cerebral embolism due to thrombosis caused directly by the administration of intracarotid therapy. At no time did I succeed in recovering either the Pregl iodine solution or the acriflavine base solution from the cerebrospinal fluid.

CASE 4.—S. G., a boy 12 years of age, was admitted to the hospital on Jan. 10, 1936. Mastoidectomy on the left was performed on Aug. 29, 1935. Although the mastoid wound healed completely, a slight mucopurulent discharge from the middle ear persisted. Two weeks previous to admission he began to complain of headache. On Jan. 3, 1936, the discharge from the middle ear became very profuse and purulent. On January 5 he began to complain of severe pain in the left eye. On January 9 paralysis of the left facial nerve suddenly appeared. The temperature ranged from 99 to 101 F. A diagnosis of suppurative apicitis was made. This diagnosis was corroborated by Dr. S. J. Kopetzky. Roentgenologic examination of the petrous pyramids was made by Dr. H. K. Taylor, who reported the existence of empyema of the apical carotid portion of the left petrous pyramid. A complete apicectomy was performed on Jan. 11, 1936. A supracochlear and infracochlear fistula was found in the basal labyrinthine portion of the petrous pyramid.

The transverse tympanic portion of the canal of the facial nerve was missing, and the exposed facial nerve was edematous and congested. The supracochlear fistula communicated with the apical carotid portion of the pars petrosa. A large pus cavity surrounded by granulations was present in the apical carotid portion. The entire outer wall of the internal carotid canal was absent. The anterosuperior wall of the middle fossa of the apical carotid portion was removed. The entire cellular structure within the apical carotid portion was exenterated. The bony wall of the posterior fossa and the inferior skull base wall were exposed and inspected. A gauze drain was inserted, and the wound was packed lightly. The mastoidotympano-apical wound was allowed to heal by granulation. Immediately after the complete apicectomy, the headache and retro-orbital pain disappeared. The paralysis of the facial nerve gradually improved and disappeared five days after the operation. The boy was discharged from the hospital on Jan. 23, 1936, in good condition.

The mastoidotympano-apical wound healed completely in six weeks. A culture of the pus from the apical abscess revealed a nonhemolytic streptococcus.

Comment.—1. In this case there existed a combination of suppurative perilabyrinthitis and suppurative apicitis. The suppurative perilabyrinthitis was undoubtedly the initial complication which was responsible for the persisting discharge from the middle ear following the mastoidectomy. This suppurative perilabyrinthitis eventually extended into and infected the apical carotid portion of the petrous pyramid, which resulted in suppurative apicitis, giving all the classic signs and symptoms of this lesion. The timely performance of a complete apicectomy undoubtedly prevented a further extension of this infection into the meninges.

2. The finding of a fistula in the basal labyrinthine portion of the petrous pyramid should be not a signal to stop but, on the contrary,

a stimulus for further exploration. Such a fistula must be considered as the outpost of a lesion situated more remotely and more deeply and must be taken as a signal to continue surgical intervention until the pathologic focus which acts as the source of supply for the pus exuding through this fistula is reached, explored and eradicated. A perilabyrinthine fistula may lead into and communicate directly with the cellular structure of the apical carotid portion of the petrous pyramid, or it may communicate with another perilabyrinthine group of cells, which in turn communicate with the apical carotid portion. If by probing a fistula in the basal labyrinthine portion one does not succeed in finding a tract leading directly into the apical carotid portion, it is no proof that the infection does not extend into it, because owing to the fact that all the perilabyrinthine cell groups are intercommunicating, a fistulous tract in one cell group may communicate first with another cell group and then in turn with the cells in the apical carotid portion. Therefore, in the presence of roentgenologic evidence of existing suppurative apicitis the finding of a fistula in the basal labyrinthine portion of the petrous pyramid must be looked on as corroborative evidence of existing apicitis, which strengthens the indications for complete apicectomy.

Case 5.—M. J., a man 64 years of age, was admitted to the hospital on May 6, 1935. The diagnosis on admission was otitis media purulenta chronica on the left, acute exacerbation, cholesteatoma and paralysis of the facial nerve on the left. He gave a history of otitis media purulenta chronica since childhood; the acute exacerbation began two weeks previous to admission with a profuse purulent discharge from the middle ear and severe headache, which persisted. Paralysis of the facial nerve made its appearance on May 5, 1935. Mastoidotympanectomy was performed on May 6, 1935.

Operation and Course.—The mastoid process was sclerotic; the mastoid antrum was filled with pus and granulations. The aditus and the tympanic cavity were filled with a macerated cholesteatomatous mass bathing in thick pus. The transverse tympanic portion of the facial canal was destroyed, with the facial nerve lying exposed, inflamed and bathing in pus. The floor of the tympanic cavity was extremely necrotic. A culture of the pus from the tympanic cavity revealed Streptococcus mucosus. After the mastoidotympanectomy the headache subsided, and the patient showed general improvement, except that the paralysis of the facial nerve persisted. He was discharged from the hospital on May 14, 1935, in apparently good condition.

On June 4, on the patient's visit to the office for dressing of the mastoid wound, a peculiar swelling was observed on the left side of the upper portion of his neck, anterior to the tip of the mastoid. This swelling increased rapidly. On June 7 an incision was made and a large dissecting collection of pus was found underneath the sternocleidomastoid muscle immediately surrounding the carotid sheath. A drain was inserted. Simultaneously with the appearance of this swelling in the neck I observed a rather profuse, purulent discharge exuding from the floor of the tympanic cavity. After the incision and drainage of the abscess in the neck, the swelling subsided, but the purulent discharge from the floor of the tympanic cavity persisted and continued to be copious. The paralysis of the facial nerve

still showed no signs of improvement. The temperature consistently stayed between 99 and 100 F. On June 12 he began to complain of headache again. On June 14 the temperature rose to 101.2 F., and he was readmitted to the hospital for observation. The spinal fluid was examined on June 16 and found normal. Headache remained his only complaint. Neurologic examination gave negative results. Examination of the spinal fluid on June 20 gave negative results. The headache persisted, and the purulent discharge from the floor of the tympanic cavity was greatly diminished. On June 25 he had a severe headache, the temperature rose to 105 F., the neck was rigid and the Kernig sign was present on both sides. Examination of the spinal fluid showed 1,200 cells and a trace of sugar and Str. mucosus was present on culture.

Complete apicectomy was performed on June 25 for the purpose of drainage of the cisterna pontis.

The entire bony floor of the tympanic cavity below the region of the promontory was missing. The jugular dome was missing, and the jugular bulb was lying uncovered, thickened and granulated. The tympanic vertical portion of the internal carotid artery was completely exposed, the external bony wall of the carotid canal completely liquefied, the internal bony carotid canal wall intact and the artery bathing in pus. Between the internal carotid artery and the jugular bulb a tract of pus was found leading downward toward the sheath of the common carotid artery. The styloid process was destroyed and absent. The entire floor of the transverse apical portion of the internal carotid canal was destroyed by the pericarotid abscess. The bony structure of the apical carotid portion of the petrous pyramid lying superior and posterior to the entire internal carotid canal was found to be sclerotic and healthy. The dura of the apical posterior fossa was exposed and incised, and the cisterna pontis was drained. The patient died on June 27, 1935.

Comment.—1. Although at no time in the entire course of the illness were signs or symptoms of suppurative apicitis observed, the headache and the sudden appearance of a profuse purulent discharge on the floor of the tympanic cavity, the origin of which could not be traced, should have led me to suspect the existence of such a condition.

2. To have suspected that the deep-seated collection of pus in the neck was the result not of an infection of some deep-seated cervical glands but rather of a pericarotid abscess dissecting its way into the common carotid sheath in the neck would have taxed my imagination too greatly, and only such observations as were made with the complete apicectomy could have convinced me of that.

3. The extremely unusual course taken by the infection of the middle ear in this case was most likely due to the fact that the mastoid and petrous portions of the temporal bone were extremely sclerotic. The jugular dome and the internal carotid canal in this case were the pathways of least resistance.

4. The inciting cause of the bacterial suppurative meningitis in this case may have been either the pericarotid abscess or the infection of the facial nerve or both. An early diagnosis of the pericarotid abscess and a timely apicectomy may have checked the extension of this infection to the meninges.

CASE 6.—W. H., a girl 6 years of age, was admitted to the hospital on March 5, 1935. The diagnosis on admission was acute suppurative meningitis. Examination of the spinal fluid showed 1,800 cells, a trace of sugar, no organism on smear and Str. haemolyticus on culture. Mastoidectomy on the right was performed two weeks previous to admission. Complete apicectomy was performed on the day of admission for the purpose of searching for a hidden focus of infection which might be acting as the inciting cause of this meningeal infection. The complete apicectomy was also performed as a necessary method of approach for surgical drainage of the cisterna pontis.

Operation and Course.—The cellular structure within the apical carotid portion was diploic. No evidence of suppuration within the apical carotid portion was discovered. The entire cellular structure was extremely congested and bleeding freely. After a complete exenteration of the apical part, the posterior wall lying anterior to the internal auditory meatus was removed, and the dura of the posterior fossa was incised infratentorially. A large amount of the infected spinal fluid escaped from the cisterna pontis; a piece of fine rubber tubing was inserted into the cisterna pontis for the purpose of maintaining drainage. The entire apical wound was packed lightly with narrow sterile gauze. The intracarotid injections of Pregl's iodine solution and acriflavine base solution were given alternately every twelve hours. The child showed no improvement, grew progressively worse and died on March 12, 1935.

CASE 7.—P. E., a woman 41 years of age, was admitted to the hospital on Aug. 29, 1935, with a history of otitis media purulenta chronica on the right of twenty years' duration. On July 4, 1935, after bathing she suffered from an acute exacerbation of the otitis media purulenta chronica on the right. The diagnosis on admission was otitis media purulenta chronica, cholesteatoma, acute exacerbation and acute suppurative mastoiditis on the right. Mastoidotympanectomy was performed on Sept. 3, 1935.

Operation and Course.—A large cholesteatomatous mass was found filling the tympanic cavity, with the aditus and the antrum bathing in pus. The dural plate forming the tympanic roof was absent, and the dura of the middle fossa was covered with granulations. Culture of pus from the mastoid process showed Str. mucosus. The headache persisted and continued to increase in severity; after the mastoidotympanectomy on Sept. 7, 1935, the patient suddenly became comatose and for the first time showed definite signs and symptoms of suppurative meningitis. An examination of the spinal fluid was done on September 7 and showed 2,800 cells, no sugar, no organisms on smear and Str. mucosus on culture. The temperature ranged between 99 and 100 F. up to September 7, when she went into coma, with a sudden rise in temperature to 103.6 F.

Complete apicectomy and drainage of the cisterna pontis were performed on Sept. 7, 1935. No focus of infection was found in the petrous pyramid. The apical carotid portion was sclerotic. The patient did not regain consciousness and died on Sept. 9, 1935.

CASE 8.—R. R., a girl 7 years of age, was admitted to the hospital on April 11, 1936. The diagnosis on admission was otitis media purulenta acuta on both sides, acute suppurative mastoiditis on both sides and acute suppurative meningitis. She had a history of otitis media purulenta acuta on both sides of three months' duration and excruciating headache for ten days previous to admission. Examination of the spinal fluid on the day of admission revealed 2,460 cells, a trace of sugar and Str. haemolyticus on smear and culture. On April 11, 1936, after a double mastoidectomy a complete apicectomy was performed on the right tem-

poral bone for the purpose of creating an approach for surgical drainage of the cisterna pontis. The posterior bony wall of the apical carotid portion of the petrous pyramid was removed, and the dura of the posterior fossa was exposed. An infratentorial incision of the dura was made, and a fine rubber tube was inserted into the cisterna pontis after the escape of spinal fluid. No pathologic process which might have been the exciting cause of the meningitis was encountered within the bony structure of the apical carotid portion or surrounding it. She showed no improvement and died on April 15, 1936.

CASE 9.—R. J., a woman 56 years of age, was admitted to the hospital on Sept. 20, 1936. The diagnosis on admission was acute suppurative mastoiditis on the right, with acute suppurative meningitis. She was brought to the hospital in an unconscious state. Examination of the spinal fluid on admission revealed 5,496 cells per cubic millimeter, with 100 per cent polymorphonuclears, and 62 mg. of sugar per hundred cubic centimeters; culture showed Pneumococcus type III. A blood count showed white cells 29,600, with polymorphonuclears 93 per cent and small lymphocytes 7 per cent. She had a history of otitis media purulenta acuta on the right of six weeks' duration. Mastoidectomy and complete apicectomy on the right were performed on Sept. 20, 1936.

Operation and Course.—A perisinuous abscess was noted in the inferoposterior perisinuous region. The sinus wall was thickened and covered with granulations. Suppuration of the perilabyrinthine cells was present. The apical carotid portion of the petrous pyramid was sclerotic; no focus of infection was found within or surrounding the apical carotid portion of the petrous pyramid. The posterior bony wall of the apical carotid portion was removed, and the dura of the posterior fossa was exposed and incised infratentorially. A large quantity of purulent spinal fluid escaped from the cisterna pontis. A drain was inserted. The patient did not regain consciousness and died on Sept. 21, 1936.

A culture of the pus obtained from the perisinuous abscess in the mastoid process resulted in a growth of Pneumococcus type III.

CONCLUSIONS

A new description and a nomenclature of the petrous portion of the temporal bone which conforms to the present day surgical requirements are suggested.

Suppurative apicitis per se is an indication for immediate surgical intervention on the petrous pyramid, as suppurative mastoiditis per se is an indication for immediate operation on the mastoid process.

A new technic is described for the surgical treatment of suppurative apicitis and its complications.

The complete apicectomy presents the following advantages:

1. It permits a thorough removal of all diseased tissue within the entire apical carotid portion of the petrous pyramid, while the apicotomy provides for puncture and drainage only.
2. It permits a thorough inspection of the posterior surface of the apical carotid portion of the pars petrosa, which heretofore has been impossible.
3. It permits complete exposure and inspection of the dura in the posterior cranial fossa anterior to the internal auditory meatus.

4. It permits inspection of the entire dura of the middle cranial fossa in apposition with the anterosuperior petrosal surface without danger of trauma to the dura proper.
5. Through the exposure afforded by this technic, an epidural abscess in the posterior fossa anterior to the internal auditory meatus can be adequately drained.
6. A collection of pus which has ruptured through the inferior surface of the apical carotid portion can also be drained. The inner table of the inferior skull base surface of the apical carotid portion can be inspected and surgically treated.
7. It will expose and drain a pericarotid collection of pus within the carotid canal.

The exposure of the internal carotid artery throughout its entire course in the bony canal is to be viewed as an advantage in guiding one safely through the entire apical carotid portion of the petrous pyramid. The internal carotid artery, because of its unvarying course, is a great natural landmark which leads from the end of the basal labyrinthine portion through the entire apical carotid portion directly to the geometric apex of the petrous pyramid and keeps one constantly in the safety zone. It is therefore almost impossible to injure it.

The facial nerve and the promontory are never in danger of injury during an apicectomy, because they both occupy an anatomic position posterior to the field of surgical instrumentation.

In all cases of otitic bacterial leptomeningitis, wherein all possible foci within the temporal bone should be removed, this procedure is suggested for the exploration of a possible hidden focus of infection within the apical carotid portion or in the regions surrounding it, which may be the inciting cause of the meningeal infection.

The complete apicectomy is suggested as a means of approach to the dura of the posterior fossa for surgical drainage of the cisterna interpeduncularis (supratentorial) and of the cisterna pontis (infratentorial) in the treatment of acute bacterial suppurative meningitis. Such drainage, if instituted early in the disease before the pathologic process envelops the whole surface of the brain, may prove beneficial in the treatment of meningitis.

The surgical treatment of thrombosis of the inferior petrosal sinus may be attempted through this route.

A complete apicectomy can be carried out when necessary in any type of petrous pyramid, pneumatic, diploic or sclerotic. Suppurative apicitis can occur in any type of petrous pyramid.

No apicectomy is complete or can be completed without complete exploration of the internal carotid artery, just as no mastoidectomy is complete or can be completed without a complete exploration of the lateral sinus.

No surgical intervention on the apical carotid portion of the petrous pyramid is adequate unless it includes the exploration and inspection of the basal labyrinthine portion of the petrous pyramid.

All surgical procedures attempted for the relief of suppurative apicitis must necessarily include a mastoidotympanectomy (radical mastoidectomy) to be adequate.

Only through a complete exenteration of the apical carotid portion of the petrous pyramid will the best results be obtained in suppurative apicitis. The complete apicectomy should therefore replace the apicotomy.

TREATMENT OF TINNITUS AURIUM BY THE INTRAVENOUS USE OF LOCAL ANESTHETIC AGENTS

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Few problems have been so elusive of solution by otologists as that of the relief or cure of tinnitus aurium. In some cases this problem has been of such magnitude as to undermine the morale of the physician as well as that of the patient. It has been felt justifiable in some instances even to sacrifice hearing by destruction of the labyrinth by surgical or other equally radical measures.

Bárány [1] discovered during the course of some intranasal surgical procedures that the injection of a solution of procaine hydrochloride into the inferior turbinate occasionally stopped tinnitus in some patients for a variable length of time. In an article published just prior to his death he suggested that this and other local anesthetic agents given by this route or, preferably, intravenously might be of some value in alleviating this condition.

This paper records experiments with three different local anesthetic agents to work out the feasibility, limitations, indications and clinical value of this type of treatment.

PHARMACOLOGY OF DRUGS USED

Procaine hydrochloride is immediately fatal to most animals when used intravenously. Fifty-five milligrams per kilogram of body weight causes convulsions and death in rabbits. It is a cardiac and respiratory depressant. It is excreted by the kidney within six hours after injection. Excretion may be hastened by the combined use of epinephrine. Death may be averted, however, even after the convulsions set in by the intravenous use of common preparations of the barbiturates.

Nupercaine is far more toxic than procaine hydrochloride.[2] The fatal dose in rabbits averages from 5 to 10 mg. per kilogram of body

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1. Bárány, Robert: Die Beeinflussung des Ohrensausens durch intravenös injizierte Lokalanästhetica: Vorläufige Mitteilung, Acta oto-laryng. **23**:201-203, 1935.

2. Lipschitz, W., and Laubender, W.: Die pharmakologischen Wirkungen des Percains: Toxikologische Untersuchungen, Klin. Wchnschr. **9**:968-974 (May 24) 1930.

weight. There is some variation from this fatal dose in dogs and cats. Death from the drug is due to paralysis of the central respiratory apparatus. The rabbit is the most sensitive of all experimental animals tested and also suffers circulatory collapse. On a rate basis smaller quantities are more rapidly fatal at a high rate of injection than larger quantities at a low rate of injection. There is apparently a slow rate of detoxication of the drug.

A combination of quinine and ethyl carbamate (urethane) is the least toxic of the three drugs used (theoretically). The lethal dose is about 326 cc. of the solution containing 1 per cent quinine hydrochloride with 0.5 per cent ethyl carbamate.

PHYSIOLOGIC EXPERIMENT

To determine the possible effect on hearing, a normal subject without tinnitus received the usual intravenous dose of the drug. Audiometric

TABLE.—*Comparative Toxicity of Drugs.* Minimal Lethal Doses per Kilogram of Body Weight in the Rabbit and Comparative Amounts of Clinical Solutions Which Would Therefore Be Expected to Be Lethal to a 70 Kg. Man*

Effective Clinical Concentrations of Solutions	M.L.D., Subcutaneous, Gm. per Kg.	M.L.D., Intravenous, Gm. per Kg.	Intravenous Expected Lethal Dose, 70 Kg. Man	Dose Used
Procaine hydrochloride 1%.....	0.175	0.030	315 cc.	7-10 cc.
Nupercaine 0.1%.....	0.2	0.002	140 cc.	3 cc.
Quinine hydrochloride 1% with ethyl carbamate 0.5%.....	.....	0.046	326 cc.	15 cc.

* Modification of table 1 in an article by N. J. Kilbourne (Local Anesthetics Producing Prolonged Analgesia: Elimination of Pain After Rectal Operation, Surg., Gynec. & Obst. 62: 599-604 [March] 1936).

readings showed no appreciable change in either the range or the intensity of hearing. Numerous patients stated that they could hear more clearly when their tinnitus was relieved, but the whispered voice tests did not substantiate the claim.

TECHNIC

At the time of the first injection numerous precautions were taken to prevent the introduction of psychogenic factors. The statement made to the patients depended somewhat on their personalities and fear of intravenous injection. These points were emphasized:

1. Whenever possible the patients were not informed that the treatment had any relation to noises in the ear.

2. When this was evident, the patient was told that this method of treatment was untried, that it was of value in a limited number of cases only and that it might even make the tinnitus worse.

Patients of all ages were included; the youngest was 17 and the oldest 81. Advanced cardiac or respiratory disease was considered a contraindication for the use of these drugs. All patients were studied for the type of deafness that they had,

if any, and other variables were noted, such as blood pressure and the sound or pitch of the tinnitus (subjective).

Intradermal cutaneous tests were made on the first group of patients treated, but this was stopped, since it was felt that it was an unwarranted assumption that patients having cutaneous sensitivity to procaine hydrochloride would necessarily have a general reaction.

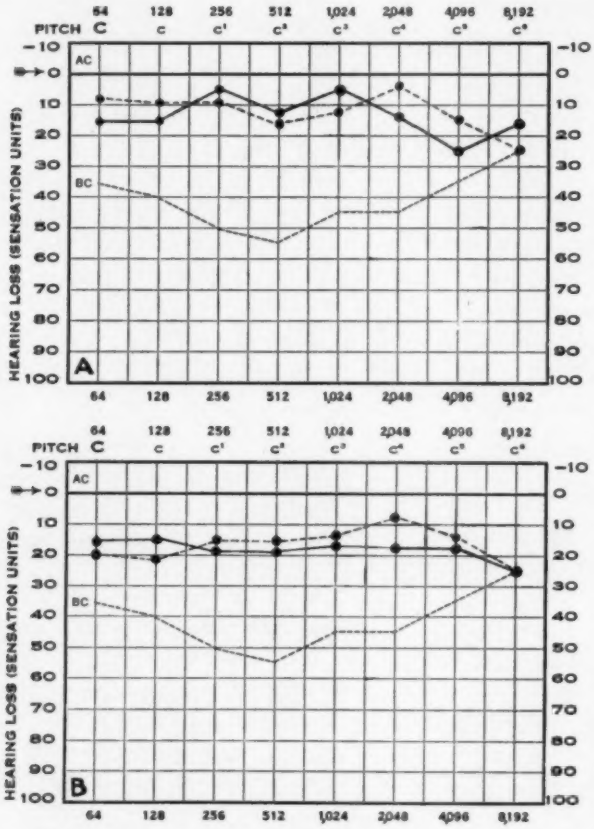

Fig. 1.—Audiograms for the same patient taken (*A*) before and (*B*) after the administration of procaine hydrochloride. The continuous line indicates the hearing for the right ear, and the broken line indicates the hearing for the left ear. The line *BC* indicates the reading of the audiometer when testing a person with normal hearing.

REACTIONS TO THE DRUGS

Procaine Hydrochloride.—In approximately 50 per cent of the patients treated there was no reaction of any kind following the injection of the drug. Of the remainder, one half, 25 per cent of the total, noticed that if they arose in less than five minutes after the injection (injections were made with the subject in the supine position) they felt slightly

dizzy; this passed away in a short time. Three patients of the series became slightly nauseated after the first injection. Two complained of feeling slightly sleepy during and for five minutes after the treatment. In general, the degree of reaction decreased with succeeding injections. There was no appreciable effect on the pulse, blood pressure or respiration.

Quinine and Ethyl Carbamate.—There were no reactions to doses of 5 cc. or less. These drugs, however, were ineffectual in doses of less than 10 cc. During injection these patients all felt hot and complained of a severe sense of constriction (cardiac ?). In one instance a patient complained of severe abdominal pain. In another marked diminution of hearing was noted, which lasted about twenty-four hours.

Nupercaine.—The reactions to this drug were exceedingly mild and paralleled those produced by procaine hydrochloride.

RESULTS

The experimental work covers a series of seventy-five subjects some of whom were observed over seven months. There was a marked variation in the response to medication, but a limited consistency corresponding to the type of tinnitus and associated deafness.

Procaine Hydrochloride.—The results are shown in the following tabulation:

1 Pulsating tinnitus not relieved in any instance (four cases)
- (*a*) Pile driver with normal hearing
- (*b*) Tubotympanic catarrh
- (*c*) Otosclerosis
- (*d*) Aortic regurgitation

2. Continuous tinnitus following trauma to the head (three cases)
- (*a*) Two cases, no relief
- (*b*) One case, 50 per cent relief, time unknown

3. Continuous tinnitus associated with otitis media (six cases)
- (*a*) Four cases, no relief
- (*b*) One case, complete relief, length of time unknown
- (*c*) One case, 50 per cent relief, length of time unknown

4 Continuous tinnitus associated with tubotympanic catarrh (six cases)
- (*a*) Three cases, no relief
- (*b*) Three cases with marked relief, one for two hours

5. Continuous tinnitus associated with normal hearing; six patients had marked relief (three listed)
- (*a*) One had marked relief for a week; after this time the tinnitus took six weeks to regain its original intensity
- (*b*) One had relief for nine weeks and now has only short attacks
- (*c*) One had relief for from five to six hours

6. Continuous tinnitus associated with mixed deafness, middle and inner ear (nine cases, five listed)
   (*a*) Effect three days
   (*b*) Effect six days
   (*c*) Effect one week
   (*d*) Effect six hours
   (*e*) Effect varied from fifteen minutes to a week
7. Deafness of the inner ear with continuous tinnitus; twenty-seven cases; results variable (six listed)
   (*a*) Marked relief with only slight buzzing remaining, effect lasted twelve weeks
   (*b*) Complete relief for three hours
   (*c*) Twenty per cent relief lasting twenty minutes
   (*d*) Complete relief for seven hours
   (*e*) Fifty per cent relief for one day
   (*f*) Failure (syphilitic?)
8. Continuous tinnitus associated with otosclerosis (five cases)
   (*a*) Effect four days
   (*b*) Pulsating tinnitus, no results
   (*c*) Complete relief for four days, better for five days
   (*d*) Marked relief averaging fifteen minutes
   (*e*) Slight relief lasted for fifteen minutes

Quinine and Ethyl Carbamate.—The results were as follows:

1. Otosclerosis; no relief, severe reaction
2. Nerve deafness; partial relief with reaction
3. Otosclerosis; complete relief for two days, severe reaction
4. Partial relief, reaction, length of time of relief unknown

Because of the severe reactions attending the use of effectual doses of these drugs, they were discontinued.

Nupercaine.—Eleven subjects were treated with nupercaine. Since it has a longer action as a local anesthetic, it was thought that it might give more prolonged relief from tinnitus and be especially indicated in cases in which procaine hydrochloride gave brief relief. The results were as follows:

1. Relief the same approximate time as procaine hydrochoride
2. Relief the same as (1)
3. No relief (average relief from procaine hydrochloride twenty minutes)
4. Four cases with relief the same as procaine hydrochloride
5. One case with relief definitely longer than procaine hydrochloride
6 Two cases with slight relief

SUMMARY AND CONCLUSIONS

Since there are so many etiologic factors in the production of tinnitus and despite this procaine hydrochloride has some effect in varying its intensity in many types, it is assumed that its action is central.

Procaine hydrochloride and nupercaine in proper doses are safe drugs to use in the treatment of tinnitus by intravenous injection.

Procaine hydrochloride is effective in reducing the intensity of tinnitus (continuous) in the large majority of cases associated with: (*a*) normal hearing, (*b*) deafness of the inner ear (only two failures), (*c*) otosclerosis and (*d*) mixed deafness.

Quinine and ethyl carbamate produce too severe a reaction to be used intravenously in the treatment of tinnitus.

Pulsating tinnitus is not relieved by this method of treatment.

Repeated doses cause no increase in tolerance to these drugs with corresponding decrease in effectiveness.

It is impossible to predict the length of time of relief of tinnitus in the average case. Its effectiveness in a case may be determined only by experiment.

THROMBOSIS OF THE SIGMOID SINUS

A CLINICAL ANALYSIS

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Septic thrombophlebitis of the sigmoid sinus remains, as it has been for many years, one of the most intriguing complications of suppurative otitis media. Instances of this condition comprise but a small portion of the relatively large number of cases of suppurative otitis media associated with sepsis. The term as used signifies an infected clot, mural or occluding, from which particles gain entrance into the blood stream and produce septicemia. True septicopyemia with a positive blood culture and metastatic abscesses is not found in every case. The broader term "sepsis" is usually applied, indicating the condition of a patient who is acutely ill of an infection and who presents chills, intermittent high fever giving the "picket fence temperature curve," leukocytosis and secondary anemia.

Having ruled out possible extra-aural causes of sepsis, one must bear in mind the fact that infecting organisms in the middle ear and mastoid may gain entrance into the blood stream without the production of a thrombus within the sigmoid sinus. As has been clearly demonstrated by Dr. Batson, of Philadelphia, there are countless small veins within the mastoid, which if infected may transmit organisms to the veins of the dura, the jugular bulb or the sigmoid sinus.

When an actual thrombus is present within the sigmoid sinus, it develops generally in one of two ways: The first is through infectious erosion of the sigmoid plate, the development of a parasinus abscess and the direct extension of infection through the parietal wall of the sinus. Second, as is frequently the case, extension to the sigmoid sinus takes place by means of thrombophlebitis of the small veins running from the middle ear and mastoid to the jugular bulb or the sigmoid sinus. In the latter way a parasinuous abscess or sinus thrombosis may be found under an intact and normal-appearing sigmoid plate.

The treatment of sinus thrombosis demands the surgeon's best judgment. Since the first operation on the sigmoid and the internal jugular vein by Lane in 1888 and that by Ballance in 1899, numerous types of surgical procedure have been recommended. These have varied from simple occlusion of the lateral sinus above the sigmoid without

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attention to the internal jugular vein or from ligation of the jugular vein alone to the radical operation on the sinus, bulb and vein as advocated by Grunert. The indications for choice of procedure do not submit readily to schematization. The decision regarding the jugular vein rests on such factors as the position and extent of the occluding or mural thrombus and the duration and severity of the patient's sepsis. In the clinic of the University of Michigan Medical School my associates and I have not found it necessary to operate on the bulb directly. Although we do not disturb the internal jugular vein as a routine in every case of sinus thrombosis, we do feel that its section and exteriorization constitute the procedure of choice for the majority of patients. In general, we have tried to be as conservative as possible in our treatment yet radical enough to remove the focus, realizing the futility of any attempt to adapt all cases of otitic sepsis to one particular form of surgical attack.

The following outline serves to propose generalizations only. The operative findings and the clinical condition of the patient should influence the surgeon to alter the outlined course in a large proportion of cases.

1. If there is discovered at the time of operation a parasinuous abscess overlying a firm, granular and apparently thrombosed sigmoid sinus, the latter is not disturbed if the patient does not demonstrate clinical evidence of sepsis. If, however, the patient should become septic in the course of a few days, an attack on the sinus becomes indicated. Such patients should have complete blood counts and blood cultures taken as a routine each day and should be observed closely for the possible development of metastatic abscesses.

2. If during the course of operation on a nonseptic patient an intrasigmoid abscess is discovered, simple evacuation and adequate drainage of this abscess may be tried as a first step, with realization of the possibility of the presence of a sterile thrombus proximal and distal to the infected portion.

3. In a septic patient with a proved thrombus in the sigmoid sinus, one must make a real attempt to carry the operation beyond the thrombus in both directions. The skull should be removed posteriorly to the midline if necessary, allowing the insertion of an aspirator into the torcular in an effort to remove the cephalic extension of the clot. If this can be done, the patient's chances for complete recovery are enhanced. Removal of a large portion of the infected parietal wall of the sinus should be accomplished in all such cases.

4. If free bleeding is obtained from both the torcular and the bulbar end of the sigmoid sinus, the internal jugular vein may be left intact, only to be severed and exteriorized if sepsis continues for from three

to five days. Despite the numerous by-paths and channels other than the internal jugular vein through which infection in the sinus and bulb can enter the general circulation, the isolation of the jugular vein remains a most valuable procedure. In several of our cases in which the vein was undisturbed at the original operation sepsis persisted for from three to six days only to subside promptly after the section of the internal jugular vein. This operation should not be abandoned, because there is no doubt as to its value in the majority of cases.

5. In general, if no bleeding is obtained from the jugular end of the sigmoid sinus in thrombosis with sepsis, the technic should be completed by sectioning the internal jugular vein between ligatures placed below the lower extremity of the thrombus and then by exteriorizing its cephalic portion. Simple ligation of the vein without its section disregards sound surgical principles by confining the infected clot to an enclosed segment and so promoting, theoretically, the chances of extension through the petrosal sinuses.

After completion of the operation on the mastoid, sigmoid sinus and jugular vein, in which one pack is placed between the skull and the parietal wall of the sigmoid sinus and another tied in the lumen of the cephalic segment of the jugular vein to control hemorrhage, the patient is given supporting treatment including small transfusions of whole blood daily or at two day intervals and the oral administration of reduced iron. All packs are left in place for five days, after which a complete dressing is done with the patient under nitrous oxide anesthesia. At this time all packs are removed and a gentle attempt is made to irrigate the isolated segment including the jugular bulb and the cephalic end of the jugular vein. Never should a forceful irrigation be given. In case hemorrhage follows removal of the packs, their reinsertion is necessary.

In the past ten years we have observed at the University Hospital fifty-two cases of thrombosis of the sigmoid sinus of otitic origin. Although this series is perhaps too small to permit the formulation of positive conclusions, there are certain outstanding points to be noted from a clinical analysis of the group. In reviewing any such series of cases, one is especially interested in determining those factors responsible for a high mortality rate. An analysis of the present series reveals the following data:

1. Three of our patients were diabetic, all of whom died.

2. There were four cases in which the thrombosis was not diagnosed but was discovered post mortem.

3. Patients suffering from sinus thrombosis complicating a chronic infection of the middle ear and the mastoid do badly.

4. Generally speaking, patients over 16 years of age carry a graver prognosis than those in a younger age group.

5. Those patients in whom the thrombosis develops late in the course of acute suppurative otitis media have a more serious outlook

TABLE 1.—*A Clinical Analysis of Fifty-Two Cases of Thrombosis of the Sigmoid Sinus*

	Recoveries	Deaths
Cases		
Number	33	19
Percentage	63.5	36.5
Age		
Under 16	25	7
Over 16	8	12
Chronic suppurative otitis media	4	8
Acute suppurative otitis media		
Less than 11 days	13	3
Longer than 11 days	16	8
Sepsis		
Less than 7 days	25	12
Longer than 7 days	8	7
Blood culture		
Positive	12	10
Negative	21	9
Type of mastoid		
Pneumatic	23	8
Diploic	5	2
Sclerotic	5	9
Type of invasion		
Erosive	14	9
Thrombophlebitic	19	10
Operation		
Ablation of the sigmoid sinus with ligation of the jugular vein	30	14
Ablation of the sigmoid sinus without ligation of the jugular vein	3	1
Extent of thrombosis		
Thrombosis discovered at autopsy	0	4
Free bleeding obtained from both the torcular and the jugular end of the sigmoid sinus, indicating a localized thrombus	15	1
Free bleeding obtained from the torcular end but not from the jugular end of the sigmoid sinus	19	9
Free bleeding obtained from the jugular end but not from the torcular end of the sigmoid sinus	0	0
No bleeding obtained from either the jugular end of the sigmoid sinus or from the torcular Herophili, but the internal jugular vein remained patent	1	1
Complete thrombosis of lateral sinus, jugular bulb and internal jugular vein	0	2

TABLE 2.—*Complications*

	Preoperative		Postoperative	
	Recoveries	Deaths	Recoveries	Deaths
Septic joints	1	1	2	0
Osteomyelitis in long bones	0	0	3	0
Pulmonary abscess	0	0	1	3
Pneumonia	0	1	2	0
Pulmonary infarct	0	0	1	0
Acute nephritis	0	0	1	0
Abscess of the brain	1	1	0	0
Meningitis	0	6	0	1
Thrombosis of the cavernous sinus	0	0	0	2
Subcutaneous abscess	1	1	2	1
Total	3	10	12	7

than do those in whom the development of the complication occurs within eleven days after the initial infection in the tympanum. Some of our patients presented diagnostic symptoms of sinus thrombosis

within from five to seven days after myringotomy. It would seem on first thought that these cases of rapidly extending, fulminating thrombosis would carry a high mortality rate. This might well be true if they were neglected. In most of the cases of this type, however, the condition developed while the patient was under close observation within the hospital, and surgical intervention was performed immediately after positive diagnosis. In practically all this group the sigmoid plate was found to be intact; in some a parasinuous abscess was found beneath the sigmoid plate; in others there was merely a gray discoloration of the dura over the sigmoid sinus suggesting the presence of a thrombus. In some of the patients who had otitis media of longer duration the advantage of making an early diagnosis was lost, since admission to the hospital came after several days of sepsis. Therein, perhaps, lies some explanation for the better prognosis noted in patients in our series who acquired sinus thrombosis early in the course of acute suppurative otitis media. In a few of the cases listed the complication might have been avoided by a timely mastoidectomy.

6. As might well be expected, there is a direct relationship between the mortality rate and the duration of the preoperative period of sepsis. From the study of our records it would seem that a very small number of patients have a chance to recover if sepsis has been present for more than seven days. This, of course, is to be expected because secondary metastatic foci have had time to develop. The mortality rate is greatly increased in those cases in which the condition has gone untreated until after dissemination of this character has taken place.

7. Those cases in which the thrombus extends into the torcular Herophili or into the internal jugular vein carry a bad prognosis.

CONCLUSIONS AND COMMENT

In contradistinction to a rather prevalent idea that the presence of sinus thrombosis is practically always an indication of a long-neglected acute suppurative mastoiditis, one must recognize that this condition may develop within the first week of otitis media by extension either to the sigmoid sinus or to the jugular bulb. In those cases of sepsis occurring during the first few days of the aural suppuration, thrombosis, if present in the sigmoid sinus, usually has developed by the thrombophlebitic route.

One should not wait until organisms are found in the blood stream before one intervenes surgically. The reports of blood cultures in the cases of this series were not uniform, and as most of the specimens taken demonstrated no growth, the importance of this procedure as a diagnostic aid is questionable. However, it is of assistance in making a prognosis, since the morbidity and mortality rates were higher in those

cases in which there were positive blood cultures. The presence of organisms in the blood stream might well mean extensive sepsis and a poor prognosis.

In every case of suppurative mastoiditis in a septic patient the sigmoid sinus should be exposed widely for examination during the operation even though the sigmoid plate is intact. Development of sinus thrombosis beneath an intact sigmoid plate by means of thrombophlebitis of the small veins of the mastoid is a common occurrence, and it may take place in a well pneumatized bone as well as in one of diploic structure. The only apparent abnormality in a thrombosed sigmoid sinus may, on occasion, be a slight gray discoloration of the parietal dural wall.

The degree of sepsis present in sinus thrombosis varies depending perhaps on the rate of entrance of bacteria into the blood stream and the bactericidal qualities of the patient's blood serum. Real shaking chills and high fever are not requisite.

There is ample time to make a diagnosis of sinus thrombosis, but this time must be utilized not only to establish a positive diagnosis but also to rule out thoroughly other possible causes of sepsis.

The prognosis for recovery from sinus thrombosis may be said to be good in patients who receive early diagnosis and adequate treatment. The mortality rate of 36.5 per cent presented here represents that found in fifty-two consecutive cases observed in the last ten years without selection. The nineteen patients who died include six presenting preoperative meningitis, while several others had experienced a prolonged period of sepsis before operation.

EXPERIMENTAL EVIDENCE FOR A BASIC THEORY OF VIBROTACTILE INTERPRETATION OF SPEECH

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THE PROBLEM

It has been demonstrated repeatedly that certain vibratory patterns of spoken language can be identified by the senses of touch and vibration. For example, one of us (R. H. G.)[1] has shown that deaf persons, or normally hearing observers having hearing eliminated, can learn to recognize vibratory patterns having the qualities of vowels and diphthongs when these patterns are communicated to the finger-tips by means of the Gault phonotactor.[2] Weichbrodt[3] has found the same thing to be true for certain consonantal qualities. Roberts[4] has found that his observers after training could discriminate between two pitches when the difference was less than a semitone and when the base was 400 double vibrations. Most significant of all is the finding[5] that two of a group of six deaf pupils (after intensive training in the use of the Gault phonotactor) were able to understand the gist of a short story by vibratory cues alone. These and many other experimental findings suggest a close relationship between the more primitive senses in the skin and the highly developed sense of hearing. In the light of the theory that the sense of touch is the phylogenetic ancestor of the sense of hearing, these experimental data suggest that Dr. Gault's success in teaching deaf persons to interpret speech by touch may be attributed to this kinship between the two senses. In other words, the sense organs

1. Gault, R. H.: On the Interpretation of Speech Sounds by Their Tactual Correlates, Ann. Otol., Rhin. & Laryng. **35**:121-136, 1926.

2. This consists of a microphone, an especially designed amplifier and a receiving unit (vibrator) by means of which the deaf are taught to interpret the vibratory patterns of speech and music thus communicated to their finger-tips.

3. Weichbrodt, M.: Tactual Compared with Visual Discrimination of Consonantal Qualities, J. Gen. Psychol. **6**:203-206, 1932.

4. Roberts, W. H.: A Two-Dimensional Analysis of Discrimination of Differences in Frequency of Vibrations by Means of the Sense of Touch, J. Franklin Inst. **213**:286-312, 1932.

5. Gault, R. H.: Progress of Experiments on the Tactual Interpretation of Oral Speech, J. Abnorm. & Social Psychol. **19**:155-159, 1924.

in the skin retain part of their primitive function and vibrations received through the skin are interpreted by the deaf observer as an auditory sensation. This view is supported by a frequent experience in the laboratory, which is called an "illusion of hearing." Often after one has experimented a short time with a normally hearing or a deafened observer, the subject will say "I believe that I hear something." Since careful experiments have failed to reveal any evidence of hearing in such cases, it seems quite possible that this is an illusion, the vibratory stimulus received through the skin being interpreted as a sound.

On the other hand, there is evidence to support the opposite view, namely, that the interpretation of speech by its vibrotactile correlates depends entirely on learning, that is, a process of attaching meaning to various vibratory patterns. In other words, it is in some respects similar to learning a code. Each vibratory pattern stands for a language symbol.

The question of the basis for vibrotactile interpretation of speech may appear unimportant and trivial to the casual reader, but in some circles it is a live issue. Many visitors in our laboratory (especially otologists and neurologists) seem quite certain that our success in getting observers to interpret speech by its tactile correlates is due to this hypothetic kinship between touch and hearing, similar probably to synesthesia. This is indeed an attractive theory. The experiment to be reported here was designed to throw light on this controversy. As will be seen, our data present clear evidence against such a proposition.

METHOD OF APPROACH

If a list of sentences is placed before an observer and the sentences are spoken into the microphone of the phonotactor, the observer can soon learn to identify each of the sentences by its gross vibratory pattern alone. Furthermore, if the observer is not given the list of sentences but is merely informed that a list of sentences is to be read over the phonotactor, he soon learns to identify the vibratory pattern of each sentence as the one occupying a particular position in the list. Thus, sentence 1 is read, and the observer is informed that he is feeling pattern 1 on the phonotactor, and so on for the remaining sentences. In this way he soon becomes able to recognize each vibratory pattern as no. 1, 2, or whichever it may be.

A method of investigating our problem is furnished by two types of learning: (1) learning to identify a vibratory pattern as a particular sentence and (2) learning to identify a vibratory pattern as one having a particular position in the list. In the second type the observer was entirely unaware of the sentence being read. He knew only that he was feeling a spoken sentence. In the first type of learning, the observer

knew the actual sentence being read. He thus learned that the gross pattern being felt represented a particular sentence as well as a particular position in a series.

The immediate problem is to determine the relative ability of observers to learn to recognize sentences by their vibratory patterns and their ability to identify each of a series of vibratory patterns by its position in the series. The problem may be stated thus: Are associations with vibratory patterns formed more easily when the required association is the actual sentence being read over the phonotactor, or when the required association is merely the position of the pattern in the list? In other words, can a series of vibratory patterns produced by speaking sentences into the microphone of the phonotactor be identified more easily than otherwise if the observer knows the actual sentence being read in a practice series? If the first hypothesis discussed is true, namely, that vibratory patterns received through the skin are directly interpreted because of the kinship between the sense of vibration and the sense of hearing, then, obviously, a series of patterns ought to be much easier to learn when the actual sentences are known. If, however, the success of the Gault phonotactor depends purely on a learning process, one should not expect to find a marked difference in the rate of learning under these two conditions.

THE STIMULUS MATERIAL

We prepared a list of ninety sentences, each of which contains exactly ten syllables and includes all phonetic elements. The sentences are presented to the observers in groups of ten. We have two forms: form A, in which the odd-numbered sentences are omitted, and form B, in which the even-numbered sentences are omitted, as illustrated in table 1. These two forms were used alternately to insure equal difficulty of the vibratory patterns represented by the actual sentences. Thus, for some observers the odd-numbered sentences are represented by pattern number only, and for other observers the even-numbered sentences are represented in this manner.

THE EXPERIMENTAL PROCEDURE

The printed list of sentences in which alternate ones have been replaced by pattern numbers only is placed in front of the observer. For example, let it be supposed that form A is being used. As sentence 1 is produced under the observer's finger-tip in the practice series, a small light beside the first sentence flashes, thus indicating to the observer that the vibratory pattern he is feeling belongs to sentence 1. Then the second sentence is produced on the phonotactor, and the light before pattern 2 flashes, thus indicating to the observer that he is feeling

TABLE 1.—*The First Ten Sentences Used in the Experiments*

The Sentences Spoken Over the Phonotactor Which Produced the Vibratory Patterns to Be Learned	The List Placed Before the Observers from Which They Identified the Vibratory Patterns Felt on the Phonotactor	
	Form A	Form B
1. Did you notice the name on the book?	1. Did you notice the name on the book?	1. (Pattern 1)
2. Yes, you may keep whichever one you please.	2. (Pattern 2)	2. Yes, you may keep whichever one you please.
3. If I had known of it, I should have gone.	3. If I had known of it, I should have gone.	3. (Pattern 3)
4. I mean to ask her if she has seen them.	4. (Pattern 4)	4. I mean to ask her if she has seen them.
5. Do you think he can earn as much as that?	5. Do you think he can earn as much as that?	5. (Pattern 5)
6. Did you affix your name to the paper?	6. (Pattern 6)	6. Did you affix your name to the paper?
7. There was no sign of rain when we left home.	7. There was no sign of rain when we left home.	7. (Pattern 7)
8. He did not learn his lesson well today.	8. (Pattern 8)	8. He did not learn his lesson well today.
9. You must return the book this afternoon.	9. You must return the book this afternoon.	9. (Pattern 9)
10. She is sure that she lost it on the way.	10. (Pattern 10)	10. She is sure that she lost it on the way.

TABLE 2.—*Summary of Data*

Observer	Number of Sentences Used	Number of Fifty Minute Periods	Percentage of Correct Identifications (Corrected for Chance) Vibratory Patterns Represented by	
			The Sentence Being Read	Pattern Number Only
T*	50	42	57	55
R	30	14	41	39
S	30	8	51	48
C	10	4	45	55
P*	90	32	60	64
A*	90	84	62	68
M	40	8	35	35
L	10	12	53	53
G	10	12	51	47
Weighted averages			56.5	58.8

* Deaf observers.

TABLE 3.—*Reliability of Data*

	Patterns Represented by	
	Sentences	Pattern Number
Number of cases	216	216
Standard deviations	9.8	12.3
Reliability of difference	1.06	
Reliability of averages	0.66	0.83
Actual difference	2.3	
Ratio of sigma difference to actual difference	2.17	
Probability of genuine difference	0.985	

the pattern of a spoken sentence which he is to identify in the future as pattern 2. This process is repeated until the list of sentences has been read through three times. The time devoted to this training is called a learning period.

A test period follows, in which a sentence selected at random is read and the observer is asked to indicate to which sentence or pattern number the vibratory pattern belongs. As soon as the response is made, the experimenter flashes the light beside the correct one and reads it again. This is done to facilitate the process of learning. The observer's responses are recorded each time, and after each sentence has been presented three times (in random order) the observer is given a rest. Both learning and test periods are repeated many times for this and the remaining eight lists of sentences.

The collection of data has covered a period of a year and a half. Three deaf and six normally hearing observers took part in the experiment, giving an aggregate of two hundred and sixteen hours. The results are summarized in tables 2 and 3.

CONCLUSIONS

The results obviously favored the hypothesis that success in interpeting spoken language by its vibratory patterns felt through the Gault phonotactor depends on learning, that is, on forming meaningful associations with the various vibratory stimuli. The somewhat significant difference in rate of learning in favor of those patterns represented merely by number may be due to an inhibitory influence of the sentences. Possibly our observers in trying to follow the pattern of a sentence gave so much attention to the individual elements of the sentence that they missed the total pattern. However, the significant thing is that the observers learned vibratory patterns when they were represented merely by number just as easily as when they were represented by the actual sentence.

Apparently, hypothetic kinship between the senses of hearing and vibration did not (as one might expect) aid our observers in identifying the patterns represented by the actual sentences. Success in identifying forms of spoken language by its vibrotactile pattern seems, as far as we have gone, to depend on learning, unaided by an original kinship between the vibrotactile and the auditory sense.

Two incidental observations resulting from this study merit comment. In the first place, this study is a demonstration, par excellence, of the effectiveness of our technic of sound elimination. The use of normally hearing observers in experiments involving the vibrotactile sense requires great precaution to avoid the operation of subliminal auditory cues and therefore is a source of great concern to every experimenter in this

field. Had our observers in this experiment received the slightest auditory cue, such as a single word or even one phonetic element, the results would have been weighted in favor of the learning situation in which the actual sentences being read were known. Since this was not the case, we feel safe in assuming that no auditory cue, not even subliminal, was present.

The other observation concerns the practical use of the Gault phonotactor in the teaching of speech to the deaf. Teachers of the deaf find the instrument useful in developing a natural flow of speech, for example, in training deaf students to speak with the conventional pattern, or melody, of speech. It has been suggested that the advantage of training with the phonotactor in this respect is made possible because before the onset of deafness the patient has learned to associate certain gross patterns with words combined to form sentences. If this were true, we should expect these associations to aid one in learning to identify vibratory patterns when the actual sentence is known. Since this has not definitely been shown to be the case, we cannot attribute a great deal of efficiency in using the phonotactor to language habits previously acquired. Consequently, training with the phonotactor should be as profitable to the person deaf since birth as to one who has become deaf later in life.

SPREADING OSTEOMYELITIS OF THE FRONTAL BONE SECONDARY TO DISEASE OF THE FRONTAL SINUS

WITH A PRELIMINARY REPORT AS TO BACTERIOLOGY AND SPECIFIC TREATMENT

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Lillie [1] in 1925 reviewed the literature on osteomyelitis of the cranial bones secondary to operations on the paranasal sinuses and reported two cases, including the observations at necropsy.

Bulson [2] in 1925 reviewed the reports of cases of osteomyelitis of the skull as a complication of frontal sinusitis which had appeared in the literature for the previous thirty years. He collected reports of fifty-five cases, with thirty-seven deaths, a mortality of 67 per cent. Further analysis of Bulson's statistics revealed twenty-eight cases of complicated chronic frontal sinusitis with a mortality of 75 per cent, and twenty-seven cases of complicated acute frontal sinusitis with a mortality of 60 per cent; twenty-six patients had osteomyelitis previous to the first operation.

In regard to the bacteria present, he reported one case in which pneumococci were found but the patient recovered, five in which streptococci were isolated and all the patients died and fourteen in which Staphylococcus pyogenes-aureus was found, with three deaths; in the last of the foregoing groups the mortality was 21 per cent.

McKenzie [3] reported his experience with five cases of spreading osteomyelitis of the skull in 1927. He was unable to account for the occasional occurrence of the disease but pointed out that it is more

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From the Section on Otolaryngology and Rhinology of the Mayo Clinic and the Division of Experimental Bacteriology of the Mayo Foundation.

1. Lillie, H. I.: Osteomyelitis of the Cranial Bones Secondary to Paranasal Sinus Operations, Ann. Otol., Rhin. & Laryng. **34**:353-365 (June) 1925.

2. Bulson, A. E., Jr.: Osteomyelitis of the Frontal Bone as a Complication of Frontal Sinusitis, Tr. Am. Acad. Ophth. **31**:102-143, 1925.

3. McKenzie, Dan: Further Observations on Spreading Osteomyelitis of the Skull, J. Laryng. & Otol. **42**:293-308 (May) 1927.

common after small than after large interventions. He said: "As to the exciting cause of the disease we are still in the dark. What it is that breaks down the resistance of the osseous tissue at one particular time is unknown. I say 'at one time' because patients will pass safely through several sinus operations and then for no discoverable reason will develop osteomyelitis after some intervention that may be quite trifling."

McKenzie pointed out that occasionally osteomyelitis is self-limiting and that in these cases the condition frequently is of the spontaneous variety. When the disease originates in the bones of the face, according to McKenzie, the tendency is for these thin flat bones to undergo necrosis *en masse* and suddenly, contrasting with the creeping or halting and leaping that characterizes the behavior of the process in the cranial vault. He stated that the simple procedure of incision and drainage, which suffices for the acute osteomyelitis encountered in the practice of general surgery, is futile when applied to spreading disease of the cranial vault, because this type of disease possesses a kind of inveterate quality by virtue of which it continues to advance either until the patient dies or until the whole of the vault of the cranium has undergone necrosis. McKenzie pointed out that the appearance of a soft, doughy swelling above one or the other of the frontal sinuses or near the middle line above the root of the nose is the first sign of beginning osteomyelitis of the cranial vault and that attempts to stop the advance of the process should be made early. He advised that a curved gutter be chiseled through the frontal bone down to the dura at about the hair line or at all events above the line of superficial edema of the skin and that the frontal bone be removed toward the diseased area in an attempt to prevent contamination.

In a later paper Watkyn-Thomas and McKenzie [4] stated that in their opinion the staphylococcus is the organism responsible for the disease as a rule. They presented for consideration the bacteriologic findings in one case, however, in which Staphylococcus aureus was cultured from the periphery of the suppurating portion, but in cultures taken from bone some distance in advance of the suppuration were a few colonies of a "coliform bacillus" and an occasional streptococcus.

Gaillard and Mounier [5] in 1926, in discussing osteomyelitis of the cranial vault, pointed out that in general there are two routes for the infection to pursue: one, by contiguity in the diploe, enclosed between the internal and the external table of the bone, a route which is more

4. Watkyn-Thomas, F. W., and McKenzie, Dan: The Bacteriology of Spreading Osteomyelitis of the Skull, J. Laryng. & Otol. **44**:253-254 (April) 1929.

5. Gaillard, René, and Mounier, P. L.: Ostéomyélite envahissante des os du crâne après les interventions pour antrites frontales suppurées, Lyon chir. **23**: 334-336 (May-June) 1926.

frequently followed in the very young; the other, by the rich venous plexuses of the vault of the cranium, in which thrombosis takes place. They expressed the belief that in the adult the venous route of propagation of the disease plays the principal part. The venous network of the vault, extremely well developed and anastomotic, is contained in the canals of Breschet and communicates above with the veins of the scalp and below with the venous system of the dura mater. It is this distribution of veins which explains the double propagation of the infection that produces subperiosteal abscess above and extradural abscess below, as well as phlebitis of the venous sinuses and encephalitis. Gaillard and Mounier expressed the opinion that in all cases in which the organism can be identified, the streptococcus, either in pure culture or associated with some other organism, can be identified, and they expressed the belief that it is the organism responsible for the disease.

Blair and Brown [6] in 1927 reviewed the literature on which McKenzie had based his first article, published in 1913, and which they stated was the basis on which the tendency of the time for radical surgical treatment was founded. They expressed the belief that they could convict McKenzie of inaccuracies in his support of his radical position. They advised against any surgical procedure except drainage of the subperiosteal abscesses as they form and reported six cases, with three recoveries. They found a streptococcus in short chains in one case, a "streptococcus" in two cases, a streptococcus associated with a staphylococcus in one and actinomycosis in one. It would seem that this last case can be properly excluded from consideration; this leaves five cases with two deaths, a mortality of 40 per cent.

Woodward [7] in 1930 expressed the opinion that in all cases the condition is attributable to infection with Staph. pyogenes-aureus. He advised making cultures in all cases of sinusitis in which operation is performed and, when a pure culture of Staph. pyogenes-aureus is found, vigorous postoperative lavage of the field with diluted solution of sodium hypochlorite.

Furstenberg [8] in 1931 published reports of a series of fourteen cases in which he had radically removed bone well beyond the apparent limits

6. Blair, V. P., and Brown, J. B.: Septic Osteomyelitis of the Bones of the Skull and Face: A Plea for Conservative Treatment, Ann. Surg. **85**:1-26 (Jan.) 1927.

7. Woodward, F. D.: Osteomyelitis of the Skull: Report of Cases Occurring as a Result of Frontal Sinus Infection with Staphylococcus Pyogenes-Aureus, J. A. M. A. **95**:927-929 (Sept. 27) 1930.

8. Furstenberg, A. C.: Osteomyelitis of the Skull: The Osteogenetic Processes in the Repair of Cranial Defects, Ann. Otol., Rhin. & Laryng. **40**:996-1012 (Dec.) 1931.

of the disease. In the group of patients operated on there were eight who recovered and six who died. Three patients who were not operated on died. In Furstenberg's discussion of the manner of advance he agreed with the views of Gaillard and Mounier. He also demonstrated that the bone tends to regenerate completely over the denuded area.

Mosher and Judd,[9] in 1933, reported their results in seven cases, in four of which the patient was well at the time of the report. Only two of the patients had been well for as long as a year, however, the others having been operated on from two to eight months previous to publication of the paper. It is generally believed that from eighteen months to two years must elapse without recurrence of the disease before the patient can be pronounced cured. Mosher and Judd found Staph. aureus present in all their cases, and Mosher, in his recent article, expressed himself as still of the opinion that Staph. aureus is the causative organism.

Mosher [10] in his paper published in 1936 did not give any mortality statistics but stated: "In reviewing all the cases which we have had at the infirmary, especially those of the last five years, our operative results have been progressively better and better in direct proportion as the operations performed were systematic and radical." Mosher stressed particularly that edema of the skin of the forehead is a rough guide to the extent of infection of the bone and periosteum. He pointed out further that if there is actual necrosis of bone, the bone would be infected without necrosis for from 1 to 1½ inches (2.5 to 3.75 cm.) beyond the necrotic portion. He advised that the whole face of the frontal bone be removed as a routine, from the hair line to the eyebrow, and that the lateral limit of the region from which bone is removed on each side be the outer angular process of the frontal bone. He advised that the bone be removed in one piece if possible.

It is obvious from the review of the literature that there is some disagreement as to the proper method of surgical procedure as well as marked divergence of opinion as to the organism responsible for the disease. There seems to be a consensus that the radical procedure gives the best results, but the mortality in any series is not much better than that of Blair and Brown, who secured three recoveries in five cases with no treatment except drainage of the abscesses as they formed in the scalp and the use of arsphenamine and blood transfusions. It might be said, however, that the group of Blair and Brown is too small to serve as the basis of an opinion in so important a matter.

9 Mosher, H. P., and Judd, D. K.: An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis, Laryngoscope **43**: 153-212 (March) 1933.

10. Mosher, H. P.: Osteomyelitis of the Frontal Bone: Notes on Three Cases, J. A. M. A. **107**:942-946 (Sept. 19) 1936.

There seems to be no way of resolving the difference of opinion as to the etiologic organism from the opinions expressed in the literature.

We and our associates at the Mayo Clinic recognize three clinical types of osteomyelitis of the frontal bone secondary to disease of the frontal sinus: the fulminating type, the localizing type and the spreading type. We recognize that there is no sharp distinction between these clinical types, one tending to shade gradually into the other, but this classification has seemed useful in deciding on the procedure to follow in an individual case. We have developed this classification in considering thirteen cases observed at the clinic. In four the involvement was of the fulminating type, and four deaths occurred, meningitis developing in from twelve to twenty-four hours after the first symptom of sinusitis. In another case, in which the condition was apparently of the fulminating type, it developed into the spreading type after simple drainage of both frontal sinuses, but the patient recovered after radical removal of the diseased bone later. There were three cases in which the condition was of the localizing type in which, after a long interval, the sequestrum was lifted out through an overlying incision. There were six cases in which the involvement was of the spreading type, with two deaths and four recoveries.

The fulminating type is the one which usually follows swimming and is characterized by intense frontal pain and generalized headache, high fever, rapidly spreading edema and a tendency to spread early to the arachnoid and cranial sinuses. In this type of case we advise small incisions over the inner angle of the brow, with a small incision into the frontal sinus to allow drainage of the pus, which is usually present under pressure. Further surgical intervention is avoided until the patient has given evidence of some power to confine or sensibly to reduce the virulence of the infection. If the patient displays no power of resistance, we believe further surgical efforts in this type of case to be futile.

In the localizing type, after an initial advance, the process becomes completely isolated by reactive barriers, with eventual marginal sclerosis of bone. In this type of case we prefer to wait until the infection has become thoroughly walled off and the diseased bone has separated as a sequestrum. An incision can then be made over the involved portion and the sequestrum lifted out.

In the spreading type there is enough resistance to prevent rapid advance but there is no tendency to complete localization. We prefer waiting a reasonable time in any case of osteomyelitis of the frontal bone to see if a tendency to localization is present. If edema of the tissues of the brow and disease of the bone as revealed by roentgenograms are of steady progress, we advise operation by the technic advo-

cated by Furstenberg. If, after an initial period of localization, the lesion again shows a tendency to advance, we believe that radical surgical intervention should be instituted. If complications, such as encephalitis or thrombosis of the longitudinal sinus, are present, we believe that the osteomyelitic bone should first be completely removed. In view of the intimate connection of the veins of the frontal bone with those of the dura it seems illogical to attempt to drain an abscess of the brain while the original focus of infection is still present and extending thrombosis of the veins of the dura is taking place. In all probability this will produce continually spreading encephalitis with meningeal irritation, which potentially may produce complicating hydrocephalus. If after the infected bone has been removed a definite fistula of the dura is found, we believe that drainage by the method of Lemaitre should be instituted. If no fistula is found, we prefer to delay drainage if possible until the superficial infection has been cleaned up. If drainage of an abscess of the frontal lobe seems imperative, it may be done through a clean field in the anterior temporal region. Usually we have found it necessary to remove the frontal sinuses completely, often with a portion of the roof of the orbit, to eradicate all the diseased bone, together with such portions of the nasal bones, nasal septum and ethmoid labyrinth as may be involved. We prefer to remove the frontal bone well beyond the apparent limits of the disease in all directions. We find the inflammatory changes in the dura a better guide to the extent to which the bone should be removed than is edema of the skin over the forehead. We prefer a cutaneous incision along both eyebrows, connected over the bridge of the nose, extending slightly beyond the external angular process of the frontal bone on both sides and then turning sharply backward and upward into the scalp. This gives free exposure of the involved sinuses as well as of the frontal bone.

The divergent opinion as to the organism which causes this type of disease seems a difficulty that should be resolved, and in an attempt to do this the following cases are presented, with the results of the bacteriologic investigation and the specific therapy developed from it.

REPORT OF CASES

CASE 1.—This patient, a man aged 22, had been operated on at the clinic for pansinusitis by the intranasal route without getting relief from a profuse, purulent nasal discharge. General physical and roentgenologic examination revealed bronchiectasis at the right cardiophrenic angle; nothing else of note was disclosed. On examination of the nose a small window to the antrum was noted on each side. There was marked polypoid change involving both middle turbinates, and the region of the spheno-ethmoid recess was filled with polypoid tissue on both sides. A fine probe could be introduced into each frontal duct, but the sphenoid ostia could not be entered because of the marked polypoid degeneration. The whole

interior of the nose was bathed in a profuse purulent secretion, which seemed to come from the frontal, ethmoid and sphenoid regions. Both maxillary sinuses also contained pus. There was deviation of the nasal septum to the left, with marked narrowing of the entire left upper pole. The remainder of the examination of the ears, nose and throat revealed nothing that had any bearing on the principal clinical problem.

This patient previously had undergone intranasal operations on the antrums and on the frontal, ethmoid and sphenoid sinuses and had received much other treatment without relief of symptoms. We thought that nothing further could be accomplished by intranasal operations. Therefore, submucous resection was performed, and a week later external fronto-ethmosphenoid sinusostomy was performed, according to the technic described previously.[11] All the sinuses were markedly diseased, and histologic study of the lining membranes removed at operation disclosed marked thickening and infiltration with polymorphonuclear leukocytes. Convalescence was uneventful, and three weeks later a similar procedure was undertaken on the left, with similar findings.

The patient's progress was entirely satisfactory until the thirteenth day after the second operation. At that time a distinct swelling was noted over the left brow, extending to the right of the median line. The patient was immediately hospitalized; both incisions were opened, and Dakin tubes were inserted into the frontal sinuses and the subcutaneous tissues. The eyelids were sutured and covered with several thicknesses of petrolatum gauze. Cultures of material taken from the wounds and the interior of the frontal sinus revealed the presence of staphylococci. A vaccine was prepared and administered. Neither flushing the wound with diluted solution of sodium hypochlorite nor the use of autogenous vaccine seemed to stop the progress of the edema, which continued to spread rather slowly. In a roentgenogram taken a week after the appearance of the edema there was evidence of osteomyelitis of the frontal and nasal bones, with most marked involvement to the left of the median line. Since the progress of the lesion was slow, it was thought best to wait to see if localization would not take place.

A month later the lesion was still slowly extending and showed a tendency to extend toward the temporal fossa on the left. It was thought that sufficient time had been allowed for localization to take place and that since localization had not occurred further delay might allow too wide extension of the lesion. Therefore, the incisions along the brow on both sides were joined across the bridge of the nose, extended beyond the external angular process laterally and then bent backward and upward to the hair line. The flap was then thrown backward over the scalp. A large portion of the frontal bone had separated as a sequestrum, and an epidural abscess was found over the left frontal lobe.

Because of the report of Watkyn-Thomas and McKenzie, the cultures were taken from the interior of the sequestrum. The frontal bone was then removed to 1½ inch (3.75 cm.) beyond any evidence of inflammation of the dura, and the wound was sutured over the brow. The lateral incisions were left open for drainage, and soft rubber tubes were placed so as to pass through and through. A transfusion of 500 cc. of blood was given in the course of the operation.

An anaerobic streptococcus was cultured, and the preparation of an antivirus was advised. While this was being prepared, 2 cc. of Rosenow's concentrated

11. Lillie, H. I., and Williams, H. L.: The External Fronto-Ethmo-Sphenoid Operation: A Critical Review of the Literature and Details of the Technic in Use at the Mayo Clinic, Minnesota Med. **18**:786-789 (Dec.) 1935.

hyperimmune polyvalent antistreptococcic serum was placed in 50 cc. of sterile physiologic solution of sodium chloride, and with this solution through and through lavage of the wound was carried out. The dressings were also soaked with this solution. In a week, when the antivirus was ready, it was used in the same manner. The response to this therapy seemed definite and marked, and the patient was dismissed in a month, with the external wounds completely healed and the

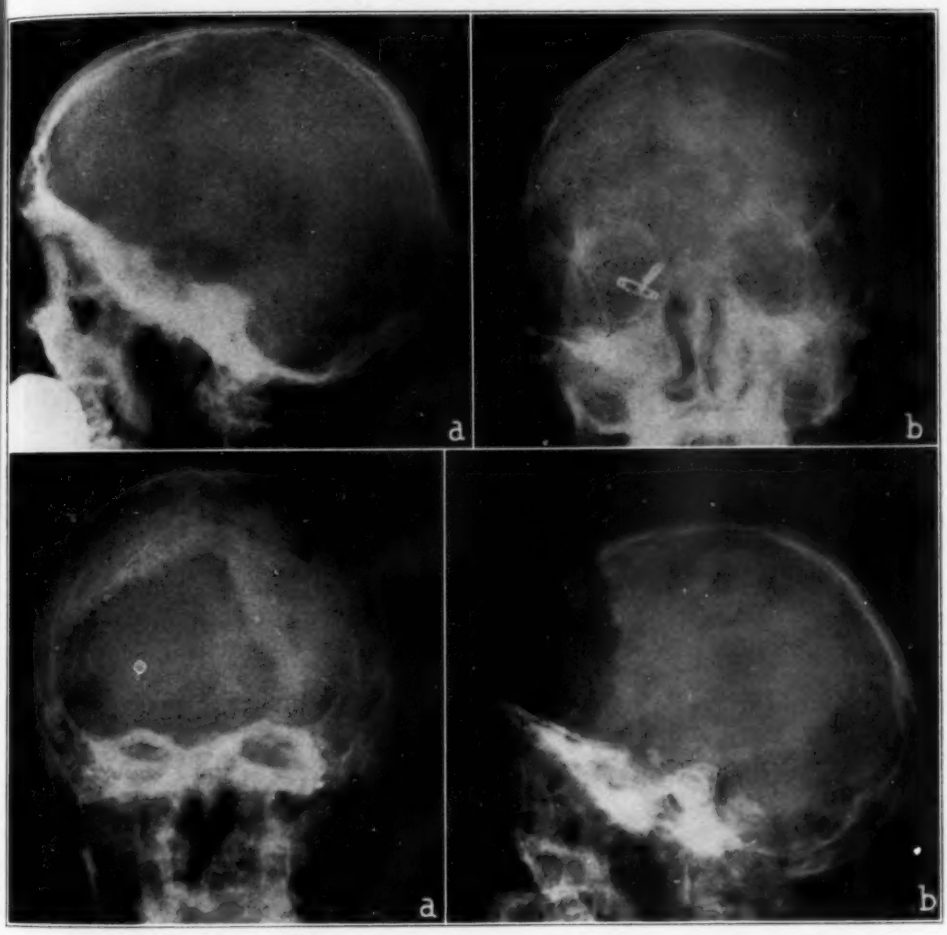


Fig. 1.—The upper figures show the skull prior to operation: *a*, lateral, and *b*, anteroposterior view. The lower figures show the area of bone removed: *a*, anteroposterior, and *b*, lateral, view.

nose free from pus. At the time of the patient's dismissal, regenerated bone could be palpated over the area from which bone had been removed, although apparently the new bone was thin. At the time of writing six months has passed since the operation without any sign of recurrence of the osteomyelitis, and the patient is free from all nasal symptoms (fig. 1).

CASE 2.—This patient, a boy aged 7 years, was brought to us for emergency treatment with the history that after a cold a month previously he had had a profuse purulent discharge from both sides of the nose. Four days before admission this discharge had stopped suddenly. On the morning of the day of admission a considerable swelling had appeared in the upper lid of the left eye, and he was brought to the clinic.

General physical examination gave essentially negative results. The right eye was normal on examination. On examination of the left eye the lids were found to be swollen shut by edema, but the globe was normal. The left pupil reacted slightly to light both directly and consensually. The left ocular fundus was normal. Hyperphoria was present on the right side, with limitation of motion of the right globe in all directions and slight displacement downward. The patient saw double in all fields, the position of the images being that characteristic of hyperphoria on the right side. It was thought that a slight degree of proptosis of the left eye was present. Examination of the nose disclosed considerable purulent discharge on the right, but the left chamber was blocked by marked swelling of the middle and inferior turbinates, and little secretion could be seen. The swelling of the left eyelid extended about 1 inch (2.5 cm.) into the forehead and across the median line at the glabella, but swelling of the lids on the right side was not noted. There was considerable tenderness over the anterior ethmoid region and over the frontal region on the left.

A diagnosis of acute ethmoiditis with rupture into the orbit was made. Since in our experience this lesion entails little danger of atrophy of the optic nerves of children and usually responds favorably to medical treatment, shrinkage and suction were advised. A roentgenogram taken at that time showed no evidence of osteomyelitis. The proptosis became gradually more marked; drainage could not be secured from the left side of the nose, and it was decided that drainage of the left frontal sinus, the ethmoid sinuses and the orbit would be necessary.

With intratracheal anesthesia the structures just named were explored. Definite evidence of osteomyelitis of the frontal bone was not found at the operation. When the periosteum was elevated over the medial wall of the orbit, a large subperiosteal abscess was found; this had formed mostly in the dome of the orbit. After the abscess cavity had been emptied, it was found that the ethmoid cells had broken through the lamina papyracea just behind the lacrimal fossa. Gas bubbles or other evidences of air or gas under pressure were not noted. The ethmoid cells were exenterated, and the floor of the frontal sinus and the fronto-ethmoid cells were removed. A large opening was made into the nose; a rubber tube drain was led from the fronto-ethmoid region out the left nostril, and the wound was closed. Proptosis of the right eye rapidly receded; however, the patient began to complain of pain in the region of the right eye, the edema over the region of the glabella increased somewhat and the discharge from the right side of the nose rapidly diminished. The symptoms and signs in reference to the right frontal sinus rapidly increased, and twelve days later an external operation was done on the right frontal sinus and the ethmoid cells. The right frontal sinus and the anterior ethmoid cells contained considerable pus, and the right frontal sinus was filled with infected hyperplastic granulation tissue. A tube was placed in the frontal sinus and was led out through the right nostril. A roentgenogram taken at that time gave evidence of slight absorption of the frontal bone in the region of the glabella. Drainage from the tubes was good, and the symptoms referable to the frontal sinus subsided; however, the patient complained of considerable frontal headache. The edema over the frontal region did not advance, and it was thought best to allow the process in the frontal bone to localize if possible.

After three weeks the patient began to have a moderately septic fever, the temperature rising to 101 F., and the headache increased. Another roentgenogram gave evidence of extension of the process in the frontal bone. Therefore, thirty days after the previous operation, the scars along the brow were reopened and joined across the bridge of the nose and the incisions were extended laterally to beyond the external angular process on each side and then were bent posteriorly into the scalp. The flap on the forehead was elevated and thrown backward over the scalp, and a subperiosteal abscess was found, with a large portion of diseased bone beneath it. The disease of the bone was more widespread than we had been led to expect from the edema over the forehead or from the appearance on the roentgenogram. The diseased bone was removed, and an epidural abscess about 5 cm. in diameter was found over each frontal lobe. The bone was removed with the rongeur to well beyond the reddened area in the dura on both sides. Two small, isolated epidural abscesses were uncovered in this process. It was necessary to remove the bone from above the hair line to the root of the zygoma together with a portion of the roof of the orbit on both sides. The wounds over the brow were closed, but the lateral wounds were left open and soft rubber tubes were run through and through for drainage. A transfusion of 300 cc. of citrated blood was given in the course of the operation.

Swabbings from the two isolated abscesses and from portions of bone removed at a distance from the region of central necrosis were cultured, and the presence of an anaerobic streptococcus which seemed identical with that recovered in the previous case was revealed. Because of the similarity of the organisms found and because of the apparently good result obtained in the first case, the same type of therapy was used, namely, lavage with Rosenow's concentrated polyvalent hyperimmune serum until an antivirus could be prepared and put to use. There was considerable secondary anemia after the operation, and two transfusions, each of 300 cc. of citrated blood, were given with an interval of a week. The wound in the forehead and the general condition showed surprisingly rapid improvement. The drainage tubes were removed in two weeks, and the patient was dismissed with the wounds completely healed and the nose free from discharge at the end of three weeks. Evidence of regeneration of bone could not be found.

At the time of writing two months has passed since the operation. In that time there has been no evidence of recurrence of the lesion and the patient has been in excellent general health (fig. 2).

METHODS OF ISOLATION OF THE ORGANISM AND PREPARATION OF THE ANTIVIRUS

The excised bone was cut into small pieces with sterile instruments, and these pieces were used for inoculation of five tall tubes of freshly made dextrose-brain broth and of some tubes of dextrose broth; the surface of blood agar plates also was inoculated. After twenty-four hours of incubation a fine cloud of growth was present at the bottom of the tubes of dextrose brain broth, and within a few hours this cloud had become diffused throughout the medium. Smears from the cultures of bone from both patients contained small, gram-positive streptococci, growing in short and in long chains. In the dextrose broth and on the blood agar plates there was no growth after four days, and the cultures were discarded. Organisms from the dextrose-brain broth grew well in subcultures in dextrose-brain broth but never could be grown in dextrose broth or on blood agar plates. The organ-

ism was thus fastidious as well as anaerobic; growth in the dextrose-brain broth was always from the bottom of the tube, where a condition of low oxygen tension prevailed.

The streptococcus was introduced at once into several 8 ounce (236 cc.) bottles of dextrose-brain broth for the production of Besredka's antivirus. After four days the contents of one of the bottles was filtered through a Berkefeld candle and was used for irrigation of the patient's wound. Two days later, when this

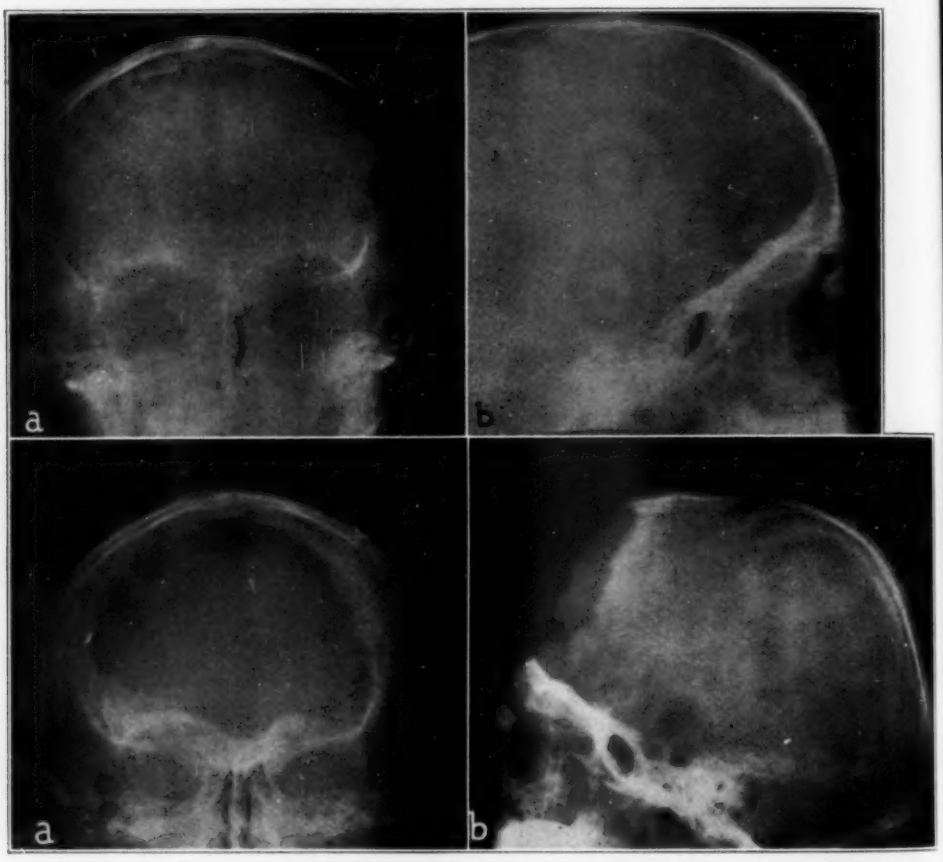


Fig. 2.—The upper figures show the skull before operation: *a*, anteroposterior, and *b*, lateral, view. The lower figures show the appearance after operation of the area from which bone was removed: *a*, anteroposterior, and *b*, lateral, view.

filtrate had been consumed, the contents of another bottle was filtered for more antivirus. Thus, every two days, another filtrate was made from one of the bottles of the original subculture. The antivirus is best when the micro-organism has grown in the medium for from twelve to fourteen days, but it can be used, if necessary, for early treatment of the patient when only four days' growth has taken place. The antivirus was supplied for irrigation until the patient's wound had so healed as to preclude further irrigations.

SUMMARY

The finding of the identical organism, an anaerobic streptococcus, in two cases of osteomyelitis of the frontal bone, together with the apparently unusually favorable results obtained by specific therapy with an autogenous antivirus, is suggestive that the organism causative of this lesion may have been isolated. This hope is intensified by the fact that previously there has not been any adequate explanation as to why in an occasional case of frontal sinusitis this lesion should develop either spontaneously or after operation, when the bone is equally exposed to infection after any type of infection of a sinus or intervention which involves it. The conjecture that the disease is of staphylococcic origin has not seemed satisfactory, because staphylococci frequently are present in sinuses which have been operated on without producing this type of pathologic change. We present our findings with the hope that they will be confirmed in further investigations by ourselves and by others. Treatment with the antivirus clinically seemed to have a favorable effect in these two cases.

We feel that delaying radical operative procedure as long as is consistent with good surgical judgment is best. We believe that this allows the natural resistance of the patient to be built up against the infection, especially as the natural resistance seems to be feeble and built up slowly in this type of case. The decision as to the optimal time for surgical intervention will be influenced considerably by the rapidity with which the inflammation spreads. In fulminating cases in which meningitis and sepsis often appear in from twelve to twenty-four hours after the first appearance of symptoms, we believe that radical surgical operation tends to hasten the spread of the disease and that only enough should be done to relieve the pressure and drain the pus that is within the frontal sinus. If the fulminating stage subsides, the surgeon should be guided by developments. We feel that the inflammatory change in the dura is the best guide to how far removal of bone should extend. It seems logical to treat the manifestations of the disease in the sinuses at the time when the diseased frontal bone is being removed. In consideration of the fact that Furstenberg and Mosher have demonstrated that propagation of the disease is by thrombosis of the dural veins which communicate with the dural sinuses and the intradural veins, it would seem best to eradicate the primary disease in the bone before attempting to treat complications, such as suppurative encephalitis or thrombosis of the dural sinuses.

Clinical Notes

AN OPERATION FOR THE CORRECTION OF ATRESIA OR STENOSIS OF THE ANTERIOR NARES

GERALD BROWN O'CONNOR, M.D., SAN FRANCISCO
Plastic Surgeon, St. Mary's Hospital

Atresia or stenosis of the nares is brought about by the lack or loss of the normal mucous membrane lining of the vestibule of the nose. In the majority of these conditions there are sufficient skin coverage and cartilage support for the tip and alae, but the lack of mucosal lining for the nasal vestibule causes complete or partial closure of the nasal apertures.

Surgical procedures directed toward the correction of atresia and stenosis of the nares must of necessity supply an epithelial lining that will restore the normal shape and contour of the nostril, nasal tip and alae, and these procedures should be so designed that postoperative collapse, shrinkage and distortion are eliminated if one wishes to restore the nose as a part of the normal respiratory apparatus.

The usual causes of atresia or stenosis of the nares, excepting the congenital abnormalities, are infection, trauma and postoperative sequelae. Among the infections, syphilis and tuberculosis are the most offending. Burns and lacerations are the most frequent of the destroying traumas. Stenosis following an operative intervention is rare, but it is occasionally laid at the door of the overzealous surgeon, who in his attempt to correct irregularity of the nasal bridge, tip, alae or nares sacrifices too much of the lining membrane of the nose or permits cicatricial contraction to occur. He must be ever on the alert to leave sufficient supporting structures and lining of the nasal tip to maintain normal air passages. Whatever the causative agent, the resulting atresia or stenosis is due to the loss of the nasal vestibular lining, and the degree of destruction or distortion of the remaining nasal structures depends on the type and severity of the destroying force. Complete atresia of the nose, whether congenital or acquired, is uncommon, so that one is usually dealing with some degree of stenosis. Stenosis occurs in the outer and upper part of the nasal vestibule. This particular fact, however, is due not to any predilection on the part of the causative agent but to the anatomic structure of the nares. The lining of the whole vestibule is usually involved, but the septal cartilage and the medial crus of the ala are rigid and give support while epithelization is taking place. The flaccid alar wing gradually contracts with the formation of scar tissue obliterating the lateral portions of the nasal vestibule. Examination in the majority of instances of stenosis of the nares reveals epithelium covering the base of the septum and the adjoining nasal floor, with stenosis occurring about the junction of the alae with the septum, the body of the ala and the base of the ala. The supporting cartilages for the ala and the tip are distorted, and the nasal skin is pulled over the free border of the ala with a rolling effect, causing an apparent or actual thickness in that area.

The operative plan to correct the aforementioned conditions is as follows: An incision is made about the stenosed airway, starting at the base of the columella, proceeding to the tip of the nose and following the natural curve of the ala. The tissue external to this incision represents the new free alar border. Sharp dissection is carried into the nasal substance, creating a pocket and exposing the medial crus of the ala, the superior ridge of the septum, the lateral crus of the ala, the lateral alar cartilage, the inferior ridge of the nasal process of the maxilla and the base of pyriform opening of the maxilla. By this dissection the distorted tip and the alar cartilages are permitted to return to their normal position; the defect is overcorrected, and a complete ring of cartilage and bone is exposed that will act as a base for the attachment of a graft, and by its rigid ringlike structure it is possible for the grafted area to shrink only to a limited degree. An impression of this subcutaneous pocket is made with Stent's composition, a dental modeling compound, and a Thiersch graft is taken, preferably from the inner surface of the arm or thigh and wrapped about the

Fig. 1.—*A*, practically complete stenosis of the left nostril following a burn of the interior of the left nasal passage by hot metal. Epithelium is present in the usual place toward the base of the septum. *B*, the nose ten days after the formation of epithelial pockets. The distorted cartilage has returned to its normal position, the contour of the alae has been improved and the columella lengthened. The thickness of the columella is due to the presence of the medial wall of the epithelial pocket. *C*, the nose thirty days after the removal of the medial wall of the epithelial pocket. The nostrils and columella are of normal size and shape, and the nasal vestibule is patent.

mold, which is inserted into the pocket. The free edges of the skin graft overlap the new border of the columella and the ala. Sutures are passed to incorporate their edges and pass over the stent impression. The whole procedure so far has been accomplished outside the nasal cavity, thus eliminating infection of the graft from nasal secretions. The mold is removed in from seven to ten days, at which time a complete graft of the cavity will have been accomplished. The same mold or its duplicate should be sterilized and reinserted daily to continue with the necessary distention of the newly lined pocket. About ten days after the epithelial pocket is made, the tissue between the old stenosed nostril and the newly grafted pocket is excised in a serrated manner, so that the medial flap of the newly epitheliated pocket can be swung into position and sutured to

the remaining mucosal lining the base of the septum and the medial aspect of the floor of the nose. The serrated incision creates a series of small flaps, which are interdigitated in a star-shaped manner with the old nasal lining to obviate the formation of a ring of scar tissue at the new juncture.

When complete atresia of the nasal passage has occurred and it is necessary to open into the nasal cavity posteriorly, excision of all the scar tissue within the nostril is essential, exposing the surrounding bony and cartilaginous structures as described. This overcorrects the original defect and permits the distorted tip and alar cartilages to return to their more normal position. An impression in dental modeling compound of this denuded area is made and covered with a Thiersch graft. A rubber tube is incorporated in the center of the mold to permit nasal drainage and respiration. The graft and mold are sutured in position anteriorly at the new openings of the nares and are held against the nasal cartilages and the bony nasal structures posteriorly. One should avoid opening into the nasal cavity at the first step if possible, for the chance of infection is materially heightened and the nasal discharge and odor are troublesome and disagreeable. When the epithelial pocket graft method is employed, one can be practically assured of a complete take in every instance.

Fig. 2.—*A*, destruction of the tip, columella, septum and lining of the nose due to syphilitic infection. *B*, the nose one week after epithelial pockets were made. The newly lined cavities, with lengthened columella and new alar wings are easily seen. *C*, the final result thirty-five days after the excision of the medial wall of the epithelial pocket. The nasal airways are patent, the left being slightly smaller owing to undercorrection of the base of the ala.

These operative procedures are preferably done with the area under local anesthesia, entailing a minimum of time spent in the hospital and of physical disability, and at the same time affording a quick and efficient means of supplying the necessary nasal lining for a permanently functioning and normal-appearing nostril.

490 Post Street

A GLARELESS EAR SPECULUM

LOUIS K. PITMAN, M.D., NEW YORK

I have found that when light is directed through the speculum now in common use, which is shaped similarly to that shown on the left in figure 1, the illumination on the objective is not uniform, spots of increased intensity of light resulting, probably due to reflection of the light rays back and forth on the curved, conical inner surface of the speculum which causes convergence of the light rays at certain spots.

A means is therefore provided to obviate this difficulty and for producing a substantially uniform light on the objective through the speculum. To this end the outer, or tip, end of the speculum is formed with a great many relatively small perforations, these being relatively close together and extending substantially half the distance from one end of the speculum to the other, or forming the entire

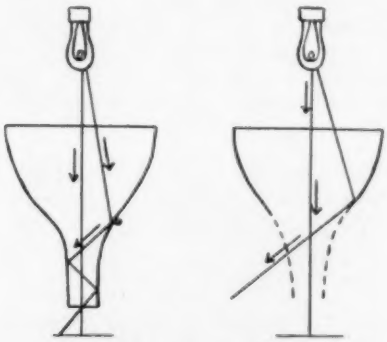


Fig. 1.—Schematic drawing showing the rays of light passing through a nonperforated and a perforated speculum. In the nonperforated speculum the light is deviated from side to side and reaches the objective from all directions. In the perforated speculum the light striking the side of the speculum is lost through perforations, and only the straight rays strike the object.

Fig. 2.—Actual photograph of the perforated speculum.

wall. I have found that with such construction a substantially uniform illumination of the objective is obtained, the reflection of light within the speculum being reduced by the perforations.

This principle for reducing the glare may be used in all other types of speculum—proctoscopes, vaginoscopes, etc.—with curved or straight sides.

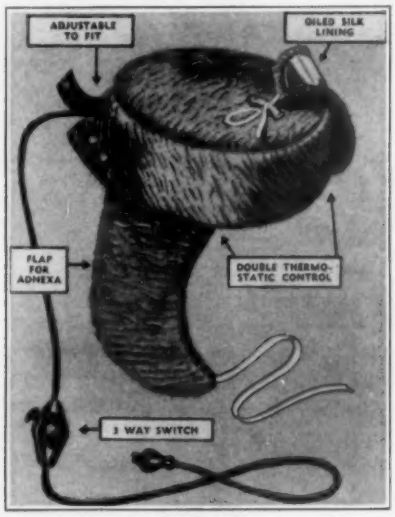
115 West Eighty-Sixth Street.

HEATING PAD FOR THE HEAD AND ADNEXA

NOAH FOX, M.D., CHICAGO

A heating pad tailored to fit the head and adnexa has been developed for persons who find it necessary to apply heat to the head but who have great difficulty in applying the ordinary heating pad to this part. Because of the circular crown, the attached flaps may easily be adjusted to any area about the head, face and neck.

The heating element consists of fine asbestos-covered nicochrome wire, mounted on a stout piece of canvas in such a way as to distribute the heat equally over



The heating pad.

the entire pad, whether it be in the crown or in the flaps. This layer is protected by a fold of heavy flannel over which is a covering of durable woolen material. It is lined with water-proof shantung silk especially prepared to resist heat. The entire fabric is easily cleansed and kept fresh with naphtha or gasoline.

There is a double thermostatic control which obviates any possibility of a burn by automatically shutting off the heat at the desired temperature. On high heat, the current is shut off at 145 F., and at low heat it is shut off at 110 F. One is thus able to keep the pad on for hours, even during sleep, without danger of a burn. The entire appliance without the cord and switch weighs 8 ounces (226.8 Gm.).

The pad is manufactured by the E. & C. Electric Company, 1716 South Michigan Boulevard, Chicago.

News and Comment

FOURTH INTERNATIONAL OTORHINOLARYNGOLOGIC CONGRESS

The fourth International Congress of Otorhinolaryngology will be held in Amsterdam, Netherlands, in 1940.

The week from July 29 to August 3 has provisionally been set for the congress.

A committee of honor has been composed by the professors of otolaryngology in the Netherlands: C. E. Benjamins, Groningen; H. Burger, Amsterdam; P. H. G. van Gilse, Leyden; P. T. L. Kan, Wassenaar; A. de Kleyn, Amsterdam; F. H. Quix, Utrecht; W. Schutter, Groningen, and H. J. L. Struycken, Breda.

The bureau of the congress consists of the following members: Prof. H. Burger, Keizersgracht 317, Amsterdam C., president; Dr. A. A. J. H. Marres, Willemsparkweg 31, Amsterdam Z., honorary secretary; Dr. M. J. ten Cate, Obrechtstraat 55, Amsterdam Z., honorary treasurer.

CORRECTION

The following footnote was inadvertently omitted from the article by Dr. R. Wallace Teed entitled "Cholesteatoma Verum Tympani: Its Relationship to the First Epibranchial Placode," which was published in the October 1936 issue of this journal:

"Part of the expenses of the present investigation were met by a grant from the Horace H. and Mary A. Rackham Fund of Detroit. Dr. D. M. Lierle, Professor and Head of the Department of Otolaryngology and Oral Surgery of the State University of Iowa, gave permission to photograph the section reproduced in figures 4 and 5. Dr. Stacy C. Howard and Miss Flora Howell, of the Staff of St. Joseph's Mercy Hospital, of Ann Arbor, Mich., gave valuable technical assistance, and Dr. A. H. Stockard, of the Department of Zoology of the University of Michigan, read the proof."

Abstracts from Current Literature

Ear

Unusually Prolonged Latency of Otogenic Abscess of Temporal Lobe with Rare Histologic Aspects. B. Kecht, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:261, 1936.

Kecht describes a large otogenic abscess of the left temporal lobe in a child aged 11. The case is worthy of attention, particularly on account of the prolonged latency, for the abscess perforated ten months after otitis media, which had lasted for about two weeks. Other unusual aspects were a deficiency of symptoms, a rare mode of transition of the causal suppuration of the middle ear —by way of dehiscences of the bones—and a rare type of membrane formation and of abscess growth. Editor's Abstract.

Resistance of the Petrosa of the New-Born to Birth Injuries. G. Kelemen, Monatschr. f. Ohrenh. **69**:1496 (Dec.) 1935.

Kelemen reports microscopic studies on the petrosas of infants who were born by means of cranial perforation. To be sure, the perforation was not the only intervention to which the craniums of these children had been subjected, for manipulation with forceps and efforts at extraction usually preceded the perforation. In the course of his studies the author found no fractures in the petrosas. The changes consisted chiefly of hemorrhages which were detected from the tympanic membrane to the internal auditory meatus, whereas the spaces of the internal ear were only slightly involved. Signs of inflammation were found widely distributed. The author thinks that if later in life auditory impairment is ascribed to trauma at birth, attention should be given to the changes that are caused by hemorrhage and inflammation of the soft parts (mucous membrane and nervous apparatus) and not so much to those in the bone tissue. To be sure, bleeding and medullary inflammation may develop in the bones in the course of birth trauma, but not fractures. Editor's Abstract.

Otomicroscopic Observations by Means of Lüscher's Instrument in Tuberculosis of the Middle Ear. E. Lüscher and S. S. Rotmann, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:10, 1935.

Lüscher and Rotmann studied fifty cases of tuberculosis of the middle ear by means of the otomicroscope of Lüscher. They observed two hitherto unknown otomicroscopic aspects of otic tuberculosis. 1. They detected small multiple nodules on a slightly inflamed tympanic membrane. This was a transitory early manifestation and was probably of a tuberculotoxic nature. This symptom disappeared without leaving traces. 2. They observed a focal, infiltrating and necrotizing tuberculosis of the tympanic membrane, which was characterized by a peculiar spotting. The spots were multiple tuberculous infiltrates, which either became caseous and thus resulted in the multiple defects of the tympanic membrane already recognized, or healed with cicatricial thickening of the tympanic membrane. The authors assert that these characteristic changes occur frequently but are detectable only with the otomicroscope. A knowledge of these changes signifies an essential progress in the early diagnosis of otic tuberculosis. Editor's Abstract.

Studies on the Cupula. W. Steinhausen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:19, 1935.

Steinhausen refutes Wittmaack's criticism of the Mach-Breuer theory and of his own studies on the cupula. He points out that the Mach-Breuer theory

assumes as cause of the irritation of the vestibular apparatus in the course of rotatory stimulation, a ring-shaped inertia current of the endolymph through the semicircular canal, the ampulla and the utricle. This ring-shaped inertia current must also attack the cupula; that is, it must produce a deviation of the cupula. The cupula is, as was formerly demonstrated by the author, larger than was at first believed. The cupula closes the ampulla practically endolymph tight. Thus, it is impossible for the current of the endolymph to pass the cupula. Consequently every current in the endolymph must produce a deviation of the cupula. According to the Mach-Breuer theory in the version of Breuer, the deviation of the cupula is the essential cause of the excitation. The Mach-Breuer theory has been thoroughly investigated. Endolymph currents were observed by Rossi and Maier-Lion, and the movements of the cupula in the course of rotatory stimulation were recorded cinematographically by Steinhausen himself. Moreover, he was able to show that the deviations of the cupula rather than the pressure produce the corresponding ocular reflexes. Thus, there can be no doubt about the correctness of the Mach-Breuer theory. Steinhausen proves further the erroneousness of Wittmaack's claims regarding the physical impossibility of the Mach-Breuer current, regarding the fixed position of the cupula, the hydrostatic stiffening and finally the stimulation of the cupula by the hydrostatic pressure. He shows that the histologic observations cited by Wittmaack as the main proof for his theories do not at all support them. The histologic method fails completely in the investigations on the intravital size and function of the cupula. Only the direct observation of the living cupula, as first accomplished by Steinhausen, has furnished definite information about the intravital size and mechanism of the cupula. Wittmaack's hydrostatic theory contradicts itself as well as everything that is known regarding the physiology of the semicircular canals, and Steinhausen thinks that for this reason it should be rejected.

EDITOR'S ABSTRACT.

EXPERIENCES IN OTONEUROLOGIC DIAGNOSIS. B. LANGENBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:63, 1935.

Langenbeck shows that the otologist has a different attitude toward the intracranial disease processes than does the neurologist. Whereas the otologist usually sees rapidly developing disorders, the neurologist is more familiar with the slowly developing brain tumor. In cases of a comparatively early, that is not yet well defined, inflammation in the region adjoining the cranial contents, the neurologist may discover numerous symptoms which are of no importance as regards the disease process on the whole. On the other hand, in cases of a severe endocranial focus of inflammation, the neurologic examination may supply no information regarding the localization. Thus, the otologist returns to the older point of view. Especially in cases of a new inflammation the neurologic examination may suggest the presence of an endocranial focus without there being any reason for alarm, since the course of the process and the patient's general condition are satisfactory. On the other hand, if general symptoms and the entire clinical aspect suggest an endocranial complication, the otologist should, of course, make use of the suggestions of the neurologist regarding localization.

EDITOR'S ABSTRACT.

POSITION AND FUNCTION OF THE LATERAL SEMICIRCULAR CANAL IN MAN AND A THEORY OF ENDOLYMPH CIRCULATION. C. F. WERNER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:194, 1936.

Werner gives his attention chiefly to the lateral semicircular canal, because this is especially important for the clinical examination of the function. He shows that in human subjects anatomic, physiologic and biologic factors do not tally with the functional predominance of the lateral semicircular canal. Studies have revealed that the plane of the lateral semicircular canal is inclined backward and downward, the angle of inclination showing considerable variation, up to 30 degrees according to some authors. Moreover, it has been observed that the

right and the left semicircular canal in the majority of cases have unequal horizontality. The author thinks that these and other variations do not correspond with the decisive rôle which has been ascribed to the semicircular canal in horizontal turning movements. He shows that anatomic, physiologic and clinical observations contradict the generally accepted opinion that in the course of the rotation reaction a current develops first in the semicircular canal and then is secondarily transmitted to the ampulla and the cupula.

On the basis of reports in the literature and of comparative anatomic studies a new theory for the solution of this problem is presented, according to which the semicircular canal, the ampulla and the utricles are regarded as parts of a closed ring. All three are of tubular structure and therefore are suited for endolymphatic disturbances. When the head is turned, the current develops in the ampulla itself. A radial position of the septum transversum and a vertical position of the axis of the crista are optimal. In this case the cupula itself is deviated by remanence. The maximum reaction during rotation is determined by the oscillation plane of the cupula and not by the semicircular canal. The latter serves for the turning aside of the endolymph and for subduing extreme currents. The plane of the semicircular canal is partly explainable by mechanical misplacement during development.

EDITOR'S ABSTRACT.

IMPROVED APPARATUS FOR DETECTING SIMULATION OF UNILATERAL DEAFNESS. F. OESTERLE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:353, 1936.

Oesterle shows that persons simulating unilateral deafness can usually be detected by the simulation test of Marx, in which the normal ear is closed by a noise drum. However, fairly intelligent patients who are anxious to obtain compensation often readily understand the nature of this test and are on guard against the unmasking of the simulation. Stenger's principle, which is based on the fact that a person with normal hearing thinks that he hears only the stronger one of two identical tones, is likewise helpful in detecting simulation of unilateral impairment of hearing; but this method too has some shortcomings. In order to overcome these, Hinsberg modified the Stenger test, and since the introduction of the electro-acoustic methods of examination further improvements have been made in this test. The device is now attached to the oto-audion models. The author describes how the test is used. He is convinced that with this improved method even the most shrewd simulator can be detected.

EDITOR'S ABSTRACT.

MEDICINAL THERAPY OF SUBJECTIVE SOUNDS IN THE EAR. H. KOESTER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:355, 1936.

Koester first presents a classification of the subjective sounds in the ear and then mentions the various disorders in which they occur. In giving his attention to the treatment, he emphasizes that the cause should always be searched for first. This is easy in some cases, but in others otologic, internal and even neurologic examination does not detect it. Moreover, even if the cause has been determined, the nature of the causal disorder is often such that effective treatment is impossible. In this connection the author mentions particularly arteriosclerosis, the baryecoia of senility, otosclerosis, chronic relapsing catarrh of the middle ear, stenosis of the auditory tubes and disorders of the tympanic membrane. In cases of deafness from toxic or medicinal causes (alcohol, quinine and so on) the causal disorder can usually be influenced by simply excluding the causal agent.

If the sounds are caused by disorders that cannot be influenced readily, the symptomatic medicinal treatment becomes important. The author mentions the various preparations that have been used for this purpose and then states that he himself uses a preparation which contains potassium bromide and a sodium barbiturate. The continued use of this medicament had been proved harmless in the treatment of chronic insomnia. It is administered in the form of tablets,

each containing 0.04 Gm. of the barbituric acid derivative and 0.6 Gm. of bromine. The tablets are dissolved in water. For the first ten days the patient receives two or three tablets daily. At the end of this period the medication is discontinued for several days in order to determine whether the sounds in the ear have been suppressed. If they recur, a smaller dose of the medicament is usually sufficient. An increase in the dosage to four tablets per day is only rarely required.

The author observed that this medicament is usually more effective in overcoming the sounds in the ear that accompany disorders of the internal ear than those that appear in disorders of the middle ear. In the majority of patients the desired results were obtained within a few days, so that the dosage could be reduced. In all, the treatment was tried in fifty cases, and in thirty-six of these the sounds disappeared. He admits that this preparation is directed only against the sounds in the ear and that the treatment of the causal disorder must be continued at the same time. However, even if the treatment is only symptomatic, it nevertheless produces gratifying results.

EDITOR'S ABSTRACT.

PNEUMATIZATION OF THE SQUAMOUS PORTION OF THE TEMPORAL BONE AS A CAUSE OF COMPLICATIONS IN ACUTE SUPPURATION OF THE MIDDLE EAR (SQUAMITIS): A CASE OF CEREBRAL ABSCESS WITH PERFORATION INTO THE VENTRICLE. K. GRAHE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:365, 1936.

According to Grahe, it is well known that the pneumatization of the mastoid process is of importance in the course of inflammation of the middle ear. Observations in recent years indicated that abnormal pneumatization of the pyramidal apex (petrositis) and of the processus zygomaticus (zygomaticitis) are of essential influence on mastoiditis. A third site at which abnormal pneumatization is likely is the squamous portion of the temporal bone. The author says that although this latter possibility has been mentioned in the literature it has not been systematically investigated.

In order to direct attention to this form of squamitis, he reports four cases in which the pneumatization of the squamous portion of the temporal bone exerted an influence on the course of acute otitis media. The first patient had acute epitympanal otitis on the right side. Roentgenoscopic examination of the mastoid process disclosed an abnormal pneumatization of the squamous portion of the temporal bone. When the mastoiditis did not subside, this roentgenologic observation induced the author to resort to antrotomy sooner than he would have otherwise, so as to avoid a complication from this source. In the second case the roentgenoscopic examination had been neglected before the first intervention and several weeks later a second operation became necessary because the squamous portion of the temporal bone with its abnormal pneumatization had become involved. The author is convinced that if the roentgenoscopic examination had been made before the first surgical intervention, the second operation could have been avoided. In the third case mastoiditis led to the involvement of the pneumatization in the squamous portion of the temporal bone. The process resulted in the breakdown of the outer table and in a subperiosteal abscess. Moreover, the dura was exposed in two places, and an extradural abscess existed. In the fourth case an abscess of the temporal lobe developed by way of the pneumatized squamous portion of the temporal lobe after suppuration of the middle ear. The abscess perforated into the ventricle, but the patient recovered.

In the summarizing discussion the author points out that the cause of the abnormal pneumatization of the squamous portion of the temporal bone is not definitely known. However, since some investigators detected pneumatization of this region frequently in anthropoid apes, the author suggests that in man it may be atavistic. He stresses that roentgenoscopic examination is a great aid in the diagnosis of involvement of a pneumatized squamous portion of the temporal bone and that it should be resorted to in all cases of otitis media. This examination and consideration of the possibility of squamitis will permit an early diagnosis and correct treatment.

EDITOR'S ABSTRACT.

Pharynx

CLINICAL CONTRIBUTIONS TO PROBLEM OF TONSILLOGENOUS SEPSIS. F. J. MAYER, Monatschr. f. Ohrenh. **69**:1415 (Dec.) 1935.

Mayer says that many years of experience and careful study indicate that surgical treatment is advisable for tonsillogenous sepsis. He mentions four phases of surgical treatment: (1) tonsillectomy, (2) opening and tamponade or drainage of the parapharyngeal and retropharyngeal spaces, (3) exposure of the vascular sheath and ligation of the jugular vein and if necessary extirpation of the glands and (4) mediastinotomy. Depending on the differences in the various cases, one or more of these procedures may be dispensed with. The author thinks that tonsillectomy is advisable in all cases. However, in those of cervical phlegmon with abscess formation, it may have to be postponed. Moreover, the order of the four phases may have to be different.

In discussing the ligation of the jugular vein, Mayer points out that except in demonstrated thrombophlebitis the value of this form of intervention is still disputed. He says that under the direction of Professor Marschik a way out is found in the form of the preliminary passing of strips of iodoform gauze under the vessel. The ligation as such is postponed until the disease process indicates it. Moreover, this preliminary passing of the gauze under and around the vessel frequently effects a partial closure of the jugular vein and thus fulfils the purpose of the ligation; yet the blood stream is not permanently interrupted. The author regards the opening of the parapharyngeal and retropharyngeal spaces as the most important phase of the surgical treatment of tonsillogenous sepsis. He thinks that this phase and tonsillectomy should never be omitted. In the concluding summary he emphasizes that surgical treatment should not be the last resort but should be instituted as early as possible. Particularly if ligation of the jugular vein is done with reservation and caution, as suggested, surgical treatment does not involve mutilation and thus should be done with less hesitation.

EDITOR'S ABSTRACT.

PATHOLOGIC ANATOMY AND CLINICAL ASPECTS OF HARD PAPILLOMA OF THE HYPOPHARYNX. F. MOSSBÖCK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:85, 1935.

Mossböck reports a case of hard papilloma of the hypopharynx, which spread to the larynx and at first presented the aspects of a malignant tumor of the hypopharynx. The absence of glandular and other metastases and the careful histologic studies on serial sections disclosed that the tumor was a so-called hard papilloma of the hypopharynx and laryngeal entrance.

In regard to the treatment the author says that radical surgical removal is advisable. From the prognostic point of view the growth is comparable with a malignant tumor, because the obstruction of the air and food passages is progressive. In the available literature the author found only one report of the occurrence of a tumor of similar structure and genesis. EDITOR'S ABSTRACT.

MENINGITIS IN ANGINA. F. JENNEMANN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:372, 1936.

According to Jennemann, meningitis is one of the comparatively rare sequels of angina. He gives detailed histories of three cases and following this discusses the pathogenesis. He points out that the spreading of the tonsillar infection takes place most frequently by way of the lymphatic system, more rarely by way of the blood stream. Once the infection has reached the parapharyngeal space, there is nothing to prevent its spreading in the direction of the base of the brain, but practical experience teaches that this possibility is rarely realized.

The treatment of postanginal meningitis consists chiefly in lumbar and suboccipital punctures and in the intravenous or intraspinal administration of methen-

amine or other substances. In the author's cases the treatment consisted chiefly of lumbar puncture, the administration of anodynes and artificial feeding. After the cerebral complications had subsided, tonsillectomy was done.

The author further reviews case histories from the literature and says that in this paper he considers only the cases of meningitis after angina and disregards the more frequently observed cases of postanginal complications, in the course of which there develop sinus thrombosis, cavernosus thrombosis, extradural abscess and other complications, with subsequent meningitis. In the summary he says that between the onset of the process, which appears harmless at first, and the development of the meningitis there elapse only a few days. There is no reliable symptom that permits the early recognition of the complication. After the treatment of the meningitis has been completed, the patient should be urged to undergo tonsillectomy. As far as the reported and reviewed cases permit a generalizing conclusion, the prognosis of the postanginal meningitis seems favorable, particularly in comparison with other forms of meningitis. EDITOR'S ABSTRACT.

Larynx

LATE NECROSIS OF LARYNX FOLLOWING ROENTGEN IRRADIATION. H. ESCHWEILER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:189, 1936.

Eschweiler reports the history of a man aged 42 who was hospitalized on account of a late roentgen-ray necrosis of the larynx, with shedding of sequestrums and dyspnea. The author feels justified in reporting this case, because the changes were especially severe and because the necrosis of the laryngeal framework developed extremely late—seventeen years after the last roentgen irradiation for lymphoma. The diagnosis of late roentgen necrosis of the laryngeal framework is justified on the basis not only of the clinical aspects but also of the preliminary history, of which detailed records were available. However, nothing definite is known regarding the doses that were applied, for in 1918 the measurement of the doses was still unreliable, so that a comparison with modern methods is hardly possible. Still, the records indicate that the first series of irradiations produced a tough subcutaneous thickening on the neck. A year later the already impaired region was again intensively irradiated from both sides, so that the larynx must have been under cross-fire. The author points out that if lymphoma of the neck is irradiated nowadays, great care is taken to exclude the larynx from the influence of the rays. He considers it surprising that in the reported case the patient had no severe complaints for seventeen years. He had only slight hoarseness and difficulty in swallowing. The hoarseness was believed to be the result of chronic laryngitis, but the author thinks that it was already the manifestation of roentgen-ray impairment of the mucous membrane. Treatment is practically ineffective, and the prognosis is doubtful in cases of this type. EDITOR'S ABSTRACT.

Nose

SERUM REACTION AS A TEST OF THYROID FUNCTION IN OZENA. W. O. RUDAKOW, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:90, 1935.

Rudakow says that studies revealed that the neutralization capacity of a serum toward atropine is proportional to the quantity of thyroid incretion in the blood. By means of a serum atropine reaction, the existence of a dysfunction of the thyroid gland could be determined, as well as the extent of the disturbance. Studies on fifty patients with ozena revealed that 50 per cent had hyperthyroidism, 32 per cent had hypothyroidism and only 18 per cent had a normal thyroid function. The author concludes that in cases of ozena with dysfunction of the thyroid gland, the latter should also be treated. EDITOR'S ABSTRACT

OPERATION ON THE MAXILLARY SINUS. W. O. RUDAKOW, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:92, 1935.

Rudakow describes an incision into the mucous membrane which he thinks is better adapted to the anatomic topographic conditions of the surgical field than those formerly used. He maintains that his method of incision involves less destruction of nerves and vessels and reduces postoperative complications.

EDITOR'S ABSTRACT.

ANATOMICOPATHOLOGIC STUDIES ON THE PATHS OF PROPAGATION OF RHINOGENIC MENINGITIS. J. JACOBSGAARD, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:387, 1936.

Jacobsgaard reemphasizes Hajek's statement that only histologic examination will demonstrate definitely the paths of propagation of rhinogenic meningitis. He thinks that the failure to realize this necessity accounts for the fact that so many of the investigations on this problem have failed to obtain reliable results. Another shortcoming of many of the studies was that they were made on patients who had undergone surgical intervention on the nasal sinuses shortly before death.

Since an operation causes considerable changes, the author decided to investigate the paths of propagation in patients who had not been operated on. He describes clinical and anatomicopathologic studies on six patients with rhinogenic meningitis. He was able to prove histologically that the lymph sheaths of the fibers of the olfactory nerve and the perforating osteomyelitis of the walls of the accessory nasal sinuses are the paths of propagation of rhinogenic meningitis. Histologic studies proved also that there are instances of epidemic meningitis in which the propagation is likewise effected by way of the lymph sheaths of the fibers of the olfactory nerve.

EDITOR'S ABSTRACT.

PYOCELE OF THE ETHMOID BONE AND OF THE FRONTAL SINUS ASSOCIATED WITH MUCOCELE OF THE OPPOSITE ETHMOID BONE. H. LEICHER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:426, 1936.

Leicher maintains that mucocele and pyocele of the ethmoid bone and of the frontal sinus are rare and that bilateral mucocele or pyocele of this region has never been reported. For this reason he feels justified in reporting a case in which, in addition to a pyocele of the left ethmoid bone and of the left frontal sinus, there developed a mucocele of the right ethmoid bone. The case is noteworthy chiefly because the development of the mucocele on one side was still in the beginning stage (mucocele of the ethmoid without protrusion into the angle of the eye), whereas on the other side it had advanced so far that the excretory duct of the frontal sinus had undergone considerable dilatation, a defect had developed in the bony floor of the frontal sinus, the eyeball had become displaced and a depression had developed in the frontal lobe of the brain. On the basis of the surgical observations it can be concluded that the mucocele originated on both sides in a dilated bulla ethmoidalis or an anterior ethmoid cell. On the left side the secondary infection of the mucocele resulted in a pyocele. The author points out that in a case of pyocele a differentiation from empyema is required. He advances evidence that in the reported case the disorder was a pyocele and not empyema, pointing out that the character of a pyocele is proved by the slow and painless development, some of the surgical aspects, the demonstration of cholesterol crystals in the cavity and the normal blood picture and speed of sedimentation.

EDITOR'S ABSTRACT.

Miscellaneous

THE DEFENSES OF THE AIR PASSAGES. S. THOMSON, J. Laryng. & Otol. **51**:1 (Jan.) 1936.

Thomson calls attention to investigations on "micro-organisms in the healthy nose," which he conducted forty years ago in collaboration with Hewlett. They

learned to appreciate that the vestibule of the nose, lined with skin and furnished with hairs and with sudoriferous and sebaceous glands, is no part of the nasal cavity proper. Micro-organisms are always abundant in it. Next, their observations demonstrated that the presence of pathogenic organisms in the interior of the nose proper must be so infrequent as to be exceptional. In 80 per cent of the observations the mucus of the nose proper was sterile. These conclusions appear to have stood the test of forty years. In this report he gives his attention to the microbial forces against which the nose, the first organ of defense, has to guard the human body.

The sterility of expired air was bacteriologically demonstrated by Gunning and by Strauss and Dubreuil. Hildebrandt showed that the air is entirely free from germs not only before arriving in the terminal alveoli but even before reaching the trachea. The author confirmed this by examining the mucus from the tracheas of animals killed in the laboratory; he always found it sterile. With Hewlett he devised an experiment which showed not only that the inspired air is sterilized before it reaches the alveoli of the lungs and before it reaches the trachea, but also that nearly all if not all the micro-organisms of the air are arrested before they reach even the nasopharynx.

The first factor of arrest is the healthy mucus. However, the mucus would remain stagnant and become useless if it were not kept constantly on the move by the action of the cilia. Cilia without their coverlet of mucus would also be no help in defense. They would dry up and cease to be. They can keep active only in a suitable moist medium.

Metchnikoff observed that nature does not use antiseptics to protect the mucous membrane; the fluids which bathe the surface of the mucosa in health are not bactericidal or are only imperfectly so. This view requires some modifying, since Fleming, in 1921, found that the nasal mucus of a person with a severe cold yields a culture in which an extraordinary bacteriolytic phenomenon is observed. The agent responsible for this bacteriolytic effect was given the name of lysozone. It is a widely distributed antibacterial ferment. At first it was thought to be peculiar to nasal mucus, but while nasal mucus and sputum are both powerful, it is most potent in tears. Linton summarizes the matter by saying: "The indications are that Lysozone plays a minor part in the resistance to invasion of the upper respiratory tract."

The author further gives a detailed description of the activity of the cilia in the different portions of the air passages and of the methods by which these were investigated and also the relations to clinical and pathologic manifestations. In the conclusion he points out that this review of the defenses of the air ways has been limited to a consideration of the local means of protection in normal conditions of health. It should not be forgotten that these are reenforced and supported, behind the front lines, by such prophylactic weapons, particularly in disease, as antitoxins and other antibodies, opsonins and phagocytosis, bacteriolysins, leukins and other germicidal substances, and all the great army of reserve defenses which maintain the normal state of immunity. These local defenses are so effective in health that, as laboratory workers know, it is difficult to produce pulmonary infection by inhalation or insufflation of organisms under normal conditions. The author's chief purpose was to show the complexity and perfection of the defenses; to appreciate their arrangement, their activities and their compensatory interactions; and to encourage interest in normal processes as a necessary first step to the study of disease.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

Forty-Second Annual Meeting, Denver, May 18, 19 and 20, 1936

THOMAS E. CARMODY, M.D., *President*

E. W. HAGENS, M.D., *Editor of Abstracts*

THE EARLY HISTORY OF OTOLARYNGOLOGY IN AMERICA WITH SPECIAL REFERENCE TO THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY. DR. THOMAS J. HARRIS, New York.

The American Ophthalmological Society was founded in 1864. At first it was thought that otologic subjects could be considered at the meetings of the society simultaneously with those having to do with ophthalmology, but it was soon found not feasible, and in 1868 the American Otological Society was founded, the first society of its kind in the world. The American Laryngological Association followed it ten years later. The American Otological Society and the American Laryngological Association, since their establishment, have served as forums for the presentation of many important contributions on laryngology and otology. Both societies were established when the number of those devoting themselves to these specialties was small. In one of them the membership was strictly limited by the constitution, and in the other it was limited by general agreement. It required, however, only a few years for the specialties to grow, and in a short time a considerable group of young men, many of whom had received their training in Europe, had established themselves in this country. The limitations to membership in the two national societies made it impossible for most of these men to become members. The need of proper connections in a professional society became more and more apparent, and after long and earnest consideration a group of those most interested came together by appointment, on June 19, 1895, at 8:30 p. m., at 25 West Thirty-sixth street, in New York, and organized a society, which was named the American Laryngological, Rhinological and Otological Society, and elected Edward Bradford Dench as president, Joseph O'Dwyer as vice president and Robert C. Myles as secretary-treasurer. There were eleven men present at this eventful organization meeting—T. Passmore Berens, Lewis A. Coffin, H. H. Curtis, Dwight L. Hubbard, Robert C. Myles, Wendell C. Phillips, J. W. Brennan, Henry Coggeshell, W. L. Culbert, N. J. Hepburn and Frank E. Miller and acceptances were received from G. E. Brewer, Brentano Clemens, L. A. Dessar, E. B. Dench, Richard Frothingham, T. J. Harris, Joseph O'Dwyer, F. J. Quinlan, Frank W. Ring, J. L. Adams and Dillon Brown. The names of most of the illustrious men in the specialty are presented, and a short discussion of their attainments is given.

STENOSIS OF THE LARYNX, WITH SPECIAL REFERENCE TO CURATIVE TREATMENT. DR. CHEVALIER JACKSON, Philadelphia.

The purpose of the paper is to present a few important points concerning stenosis of the larynx. Stenosis in the new-born is discussed, as are syphilitic stenosis and postcancerous stenosis.

The best method of treatment of laryngeal stenosis is by dilation by pressure from an elastic device, preferably a special molded rubber dilator that is shaped to correspond to the lumen and orifice of a normal larynx and that has a retaining swell to dispense with strings, wires and other kinds of mechanical devices for

fixation. Moreover, this avoids the risk of septic and pulmonary infection by permitting daily changes of the cannula and biweekly changes of the rubber.

Because of the aid obtainable by forced growth, children should not be allowed to grow up as hopeless wearers of cannulas. Ninety-five per cent of such patients can be cured by patient, careful, painstaking work.

If there has been great loss of cartilage, transplants naturalized in tongued flaps may be necessary in rare instances. Usually vestigial remains of cartilage suffice when the elastic rubber dilators are used.

DISCUSSION

Dr. Thomas E. Carmody, Denver: In the treatment of this condition Dr. Jackson has seen hundreds of patients, while most physicians have seen a single patient. One should be careful not to overtreat the developing larynx and the developing cartilage. I have used the old method of dilation. Dr. Jackson's method is so much in advance of this that in another case I should certainly use it. A larynx showing symptoms of acute tuberculosis should not be dilated.

Dr. Robert Levy, Denver: I have always been conservative in the treatment of laryngeal tuberculosis. Today the use of the galvanic cautery is the most radical treatment. The young woman I presented is better off with a tracheotomy tube than she would be if I had undertaken any surgical procedure in the larynx and perhaps activated the tuberculous condition. Persons with a tracheotomy tube may live happily for years.

Dr. Frank R. Spencer, Boulder, Colo.: There are several types of lesion which produce obstruction or stenosis in the larynx, for example, syphilis and pseudo-edema. I believe that the method of gradual dilation which Dr. Jackson has shown is much more desirable than the older methods of overcoming the cicatricial contraction.

Dr. Sidney Israel, Houston, Texas: Without any knowledge of what kind of rubber intubation tubes or core models Dr. Jackson has had made, I devised a method of attaching a rubber tube to the familiar Jackson tracheotomy tube, with a vertical post. I use a nasopharyngoscope through the tracheotomy wound to see the lesion from below. I think that the intubation tube of Dr. Jackson probably will be of advantage. However, the rubber tubing attached to the vertical post has been satisfactory and does not become detached.

Dr. Julius P. Dworetzky, Liberty, N. Y.: In treating tuberculosis one should concentrate on the treatment of the pulmonary condition. Conservatism is the main point in the treatment of tuberculosis of the larynx.

Pulmonary Collapse Following Tonsillectomy Under Local Anesthesia: Report of a Case. Dr. Samuel Iglauer, Cincinnati.

The various etiologic factors mentioned in the literature are reviewed. So far as I know, no similar case has been reported. The patient was a man 18 years old. After removal of the tonsils his temperature rose to 104.8 F., the pulse rate to 120 and the respiratory rate to 30. The next morning he complained of pain in the chest and general discomfort. The roentgen and physical examination showed atelectasis of the right side. At bronchoscopy 3 cc. of mucopurulent material was removed from the right bronchus. After bronchoscopic examination the patient continued to expectorate blood-stained mucopurulent material. However, roentgen and physical examination showed rapid recovery, and the patient was discharged ten days after tonsillectomy.

The question is raised as to the possible relation between atelectasis and post-tonsillectomy pulmonary abscess.

DISCUSSION

Dr. I. D. Kelley Jr., St. Louis: In 1890 Pasteur said that massive collapse is due to pressure exerted by collapse of the chest wall and elevation of the diaphragm resulting from paralysis of these structures. Briscoe expressed the

belief that the condition is due to inflammation affecting the muscles of the crus situated behind the peritoneum and throwing out of action one half of the diaphragm. Jackson expressed the belief that obstruction of the bronchioles is a cause of pulmonary collapse. Bradford stated that some spastic reflex of the bronchioles causes them to be occluded, permitting the subsequent absorption of air from the segment of the lung involved. According to Sante's analysis, the most plausible explanation is that of Bradford. In searching for the cause in this case, can one find its explanation in any of the four theories, or is it simply an uncomplicated inflammatory extension from a pulmonary abscess, as Dr. Iglauer suggests?

Dr. Louis H. Clerf, Philadelphia: I think that the majority of bronchoscopists are in agreement that obstruction plays an important part, if not the entire rôle, in this condition. In a case which I recently observed blood clots were noted in the left bronchus after tonsillectomy. It was found that the patient was tuberculous and had had a pulmonary hemorrhage.

Basaloma, or So-Called Cylindroma, of the Air Passages. Dr. Joseph C. Beck and Dr. M. Reese Guttman, Chicago.

There is a group of rare tumors occurring in the mucosa of the respiratory tract and its adnexa, which present a typical and characteristic clinical course and unique histologic architecture. While in the past there has been some disagreement as to the histogenesis and classification of these neoplasms, it is probable that they are mucosal basal cell growths and show a biologic course comparable to that of basal cell tumors originating in the skin. The term cylindroma is descriptive of the peculiar morphology of these neoplasms, but they are probably more correctly termed cylindromatous basalomas. Histologically they present a picture of cell nests surrounded by a connective tissue stroma. The cell nests frequently show a central lumen containing pink-staining material and sometimes cellular débris. Clinically, like their counterparts of the skin, they are slow growing, locally destructive and not metastasizing, and they recur after repeated removal. They are relatively radiosensitive and are possibly best treated by extensive resection or by adequate irradiation. Three tumors, one originating in the antrum, one in the sphenoid sinus and one in the trachea, are described, and the reports of the cases are added to those of 37 similar ones in the literature.

DISCUSSION

Dr. Chevalier Jackson, Philadelphia: The experience of Dr. Beck and Dr. Guttman has been corroborated to an extent sufficient to warrant choosing irradiation in preference to operation in these cases. One matter not touched on by the authors is the differences among histologists as to the classification of tumors other than squamous cell epithelioma. I should like to ask Dr. Beck if in his case of tracheal basaloma there was any reason to suppose that the growth started in aberrant thyroid gland tissue. In one of my cases that seemed to have been the point of origin.

Dr. Samuel Salinger, Chicago: In this condition the trouble has been that the tumor was named from its morphologic appearance rather than from its histologic genesis. It was only when Ribbert and Krompecher, especially the latter, saw more clearly that these tumors bear a strong resemblance to basal cell carcinoma of the skin that light began to appear. The weight of evidence seems to point to the fact that the basal cells originating from columnar epithelium often manifest atavistic tendencies whereby they are able to produce a secretion which is responsible in part for the cystlike structure of the growth. Histologically these tumors are at times difficult to distinguish from transitional cell carcinoma. The latter, however, develops quickly, the original focus being frequently overshadowed by early and extensive metastases, and shows a high degree of malignancy throughout. The two types, nevertheless, are both radiosensitive. Basaloma in its early stages is benign and should be amenable to radical surgical treatment.

DR. M. R. GUTTMAN, Chicago: The pathologist who examined the biopsy specimen does not believe that the growth on the trachea was a portion of thyroid tissue.

THE AUTONOMIC NERVOUS SYSTEM: SOME FUNDAMENTALS FOR OTOLARYNGOLOGISTS. DR. DAVID HIGBEE, San Diego, Calif.

In this brief review of the anatomy and physiology of the cranial autonomic nervous system some statements are made which are contrary to the prevailing opinions. I have ventured to differ from them by selecting certain known facts of anatomy and physiology which seem to give a clearer, more logical and more probable explanation of the pain which has hitherto been attributed to the sphenopalatine ganglion and the vidian nerve. This concept is in keeping with the idea of a fundamental division of the nervous system into two functional systems, viz., sensorimotor and autonomic, in which each has its definite rôle. It attributes pain to the sensory division, viz., the fifth, seventh, ninth and tenth nerves, which are known to have receptors for that sensation, and it leaves uncompromised the logical conclusion that the autonomic system is efferent, distributed to glands, smooth muscle and blood vessels, and is concerned only with vegetative functions.

DISCUSSION

DR. HARRINGTON B. GRAHAM, San Francisco: The crux of the situation, as far as this paper is concerned, is the last statement, in which Dr. Higbee assigns a lack of appreciation of pain to the autonomic nervous system. I think that hardly any one can subscribe to this supposition, as all the work that has been done along the line of relief of pain in the viscera has been based on the fact that the autonomic nervous system together with all the other portions of the nervous system is capable of appreciating pain. For instance, the pain in the uterus that accompanies carcinoma has been relieved by stripping the sympathetic nerves from the carotid artery. The recent work of all surgeons in relieving all kinds of pain in the abdomen through interference with the sympathetic nervous system would indicate that the sympathetic nervous system has something to do with the transmission of pain. This applies not to one branch of the system but to both branches, and it has been stated that any form of nervous tissue is capable of appreciating pain. The only question is how the exciting factor is produced.

DR. MERRILL C. JOBE, Denver: I think that Dr. Graham has expressed in the correct manner the inability of those present to subscribe to the conclusions of Dr. Higbee's paper. During the war Leriche relieved soldiers of terrible pain from gunshots through the brachial plexus and the median nerve by stripping the sympathetic nervous fibers going along both the artery and the median nerve. The sympathetic nervous system is so complicated that I do not believe that one can be didactic about it and decide that no pain fibers are supplied through the afferent nervous system.

DR. EDWARD C. SEWALL, San Francisco: I am not convinced that, as Dr. Higbee has said, these patients always present a psychologic factor that is not understood and that one can never understand them. I have removed the sphenopalatine ganglion through an antrum approach, but I cannot say that I have learned much as to this question.

DR. DAVID R. HIGBEE, San Diego, Calif.: I have carried on correspondence with Drs. Cannon, Dandy, Moore and Loyal Davis, and I have received their endorsement.

VASOMOTOR RHINITIS FROM AN INTERNIST'S POINT OF VIEW. DR. T. D. CUNNINGHAM and DR. J. C. MENDENHALL, Denver.

It may be said that vasomotor rhinitis is apparently a symptom of some local or remote disturbance in the functioning of the body, due to allergy, infection, fatigue, lack of vitamins or gastro-intestinal disturbance, and the treatment becomes a problem of research in each case until the causative factor or factors

have been eliminated and the local condition resulting from these factors has been remedied.

DISCUSSION

DR. DUNBAR ROY, Atlanta, Ga.: I think that this condition must be managed by a group of specialists, but should be absolutely in charge of a rhinologist with an unbiased sense of observation, coupled with a horizon which looks beyond the field of the nasal cavities. Benefit to vasomotor rhinitis is produced in many cases just as effectively by local applications as by systemic regulations, but with the combination of the two one can certainly feel that one has accomplished the best for the patient.

DR. HARRY L. BAUM, Denver: I concur in almost all that Dr. Cunningham has said. However, the therapeutic results are not always what one might wish, probably because there are more factors involved than one has any way of knowing. There is a question in my mind as to whether every rhinologist should not undertake the diagnosis of this condition, because by careful laboratory study and by following it through he acquaints himself much better with the conditions with which he deals.

DR. JOHN G. MCLAURIN, Dallas, Texas: In these cases I think that what is really meant by "heredity" is a sensitive autonomic nervous system. The question comes up as to how one can alter the sensitiveness of the autonomic nervous system. I believe that the system is under the control of the endocrine glandular balance, which in turn is probably dependent entirely on alterations in the chemistry of the body. If one is to do patients with this condition any good, one must try to work out a matter of body chemistry with them, and though that plan of procedure may not bring quick results, it will in the long run bring the best results. One may be able to get immediate results and to protect the patient from the reactions by some local treatment, by some form of medication or by the introduction of some hormone which that person lacks.

DR. THOMAS J. GALLAHER, Denver: I wish to call attention to one fact, which is that the chemistry of the blood is habitually ignored in many cases. The correction of the endocrine imbalance is essential.

DR. LOUIS LEVY, Memphis, Tenn.: The thing I want to bring to attention particularly in this work is that before one starts even a scratch test in these cases one must work with the internist and others.

TREATMENT OF ANGIOMA OF THE FACE. DR. FREDERICK A. FIGI, Rochester, Minn. This paper was published in full in a previous issue of the ARCHIVES (**24**:271-281 [Sept.] 1936).

DISCUSSION

DR. ARTHUR J. MARKLEY, Denver: Dr. Figi has referred to the fact that each worker in this field usually limits himself to one or at most two methods of treatment. In my experience the two have been implantation of radium and electrocoagulation. In cavernous angioma the irradiation required should be obtained in the shortest time possible, and the radium should be implanted deep in the lesion to spare the skin and the underlying immature tissues. The compact angioma is usually sharply circumscribed and lies close to the surface of the skin and fortunately, being radiosensitive, may be satisfactorily dealt with by filtered radiation from radium at a short distance. Damage to the skin has to be accepted as inevitable in these cases.

DR. LEROY A. SCHALL, Boston: I wish to ask Dr. Figi about a youth of 20 who has had a cavernous hemangioma of the pharynx. I removed it after ligating the carotid artery, but at the present time, one month after removal, the tumor is about 40 per cent of its former size. I should like to know whether Dr. Figi would recommend its further destruction by electrocoagulation.

DR. FREDERICK A. FIGI, Rochester, Minn.: Pertaining to Dr. Schall's case, I should continue with intensive irradiation, and if there were no response, I should expose the growth and use electrocoagulation gingerly.

FIFTEEN YEARS' EXPERIENCE WITH DRAINAGE TUBES AFTER ANTROSTOMY IN CHILDREN. JOHN J. SHEA, Memphis, Tenn.

If the child's condition warrants its use, ether is the best anesthetic, because the operative procedure can be deliberate and thorough. In the cases in which the kidney is involved either nitrous oxide or an ethylene mixture is safer than ether, even though the operation is hurried.

The nares are cleansed by suction, and the site of operation is painted with a 30 per cent solution of alcohol. The ostium of the antrum is sought and dilated. If it is not found, the membranous tissue of the middle meatus is perforated. The success of the operation depends on maintaining the patency of the ostium. Some of the earlier failures were the result of not appreciating the necessity of this second opening. A culture is made of material from the antrum. This is done through a straight trocar, with a syringe and a needle. If the patient is suspected of being allergic, a sterile physiologic solution of sodium chloride is used; otherwise brain broth medium is instilled and withdrawn. At the height of the phage craze, it was thought that leaving some of this medium within the antrum was of value, utilizing the sinus as an incubator for the growth of the phage, thus helping auto-immunization.

The naso-antral window is made with an antral trocar in the younger children and with a curved gouge in the older children. The aperture is then shaped to fit the rubber tube by a series of dilations with Ritter sounds. The catheter is then inserted, one of the smaller Ritter sounds being used as an obturator. There is a characteristic feel when the catheter slips through the window. Suction is then applied to the drain. The appliance used consists of a medicine dropper, which visualizes the contents of the antrum. It is of value to note that air bubbles are freely mixed with the contents of the antrum, for these bubbles denote the patency of the secondary opening. If no bubbles are visible, the solution of sodium chloride is forced through the tube until it appears in the back of the nose of the nasopharynx, thus blowing out any plug that might be blocking the ostium. Suction is again tried, and if no bubbles are visible through the glass, the tube is removed and the steps of the operation are repeated, special attention being given to the ostium.

This has been simplified to keeping the tubes open and the instillation of aphenolated nasal oil about the tubes. Acetylsalicylic acid is given at regular intervals according to the age of the patient, for it requires less to prevent a headache than to stop one. Many of the popular chemical antiseptics have been tried for irrigation of the antrum, but they were discarded as of no particular value.

On the fourth morning the tubes are removed by instilling a solution of sodium chloride through them, which aids in their expulsion. So far, the treatment has been painless, and the child should not be afraid of the surgeon. The irrigations are continued until the discharge has ceased and may be reestablished at any time if the drainage is impeded or if the opening has closed. The children are watched for a long time, and on the return of symptoms of sinusitis irrigation is repeated. In fact, it is advisable to irrigate these antrums at the end of any acute infection of the sinuses, remembering that it is not the amount of discharge from the sinus but the lack of drainage, with absorption of the discharge, that harms the child. The ostium of a child's antrum is relatively larger than that of an adult, and with a small cannula it can be negotiated and the antrum readily irrigated.

The preparation of the autogenous vaccine requires a week, and during that time some form of antigen or protein therapy is administered to increase the immunity, thus assisting the sinuses in their fight against the infection. The autogenous vaccine is given consistently; one series of treatments in the fall, another in the winter and a third in the early spring, with sustaining doses whenever acute sinusitis is threatened. When the pathologic condition has been thoroughly established in a child's sinuses, it requires one or two years of constant attention to reestablish the normal functions.

DISCUSSION

DR. H. W. LYMAN, St. Louis: In the St. Louis Children's Hospital a number of years ago my associates and I were enthusiastic about the use of rubber drainage

tubes in the antrum. However, in a good many of the cases we found that there was a reaction. After investigating this condition, we found that boiling the tubes or soaking them in an alkaline solution for forty-eight hours did away with the possibility of irritation. From a practical point of view the tubes are pleasing. I think that there is some risk involved in enlarging the natural ostium in young children. We have not found it necessary to do this.

Dr. Theron E. Fuller, Texarkana, Texas: In our cases my associates and I have packed the nose with a hypotonic solution of sodium chloride and with a 2 per cent solution of phenylamino-ethanol sulfate, which is prepared at the laboratory of Dr. George Piness of Los Angeles. The infra-red light is also useful. In severe cases blood transfusions are better than streptococcic antigen. I have come to rely largely on suction by the Proetz displacement method, using a 0.5 per cent solution of ephedrine in a physiologic solution of sodium chloride.

Dr. J. B. Nail, Wichita Falls, Texas: I have used anesthesia induced by n-methylcyclohexamylmethylmalonylurea 100 times in children and like it. After losing track of a tube in one case, I have retained the tubes in place by the use of adhesive and a silk suture or, if the involvement is bilateral, by sewing the tubes together.

Dr. John G. McLaurin, Dallas, Texas: In my experience, retention tubes have been irritating and straight tubes became loose. If the windows are made large enough, and postoperative irrigations are given, they will remain open. For irrigation a solution of sodium chloride is the one of choice.

Dr. John J. Shea, Memphis, Tenn.: I think that the middle meatus in all children is the easiest approach to the antrum.

The Antrum Operation: A Method Affording Permanent, Adequate Drainage When Cure of the Empyema is Essential. Dr. Edward Cecil Sewall, San Francisco.

Earlier operations on the antrum are discussed. The Caldwell-Luc technic was a definite improvement; yet grave consequences sometimes follow the operation. In the case reported here the antrums had previously been operated on by the Caldwell-Luc method. Because of the badly diseased condition of the antrums, I reoperated. Healing failed to take place, and at the next operation a large opening was left at the canine fossa on both sides. One side closed again, and a final operation was performed. The opening in the region of the canine fossa is kept open by a self-retaining special rubber tube. My associates and I have now operated on 14 patients by this method and are pleased with the results.

DISCUSSION

Dr. David R. Higbee, San Diego, Calif.: These cases represent those in which the ordinary procedures failed and heroic measures were used, and by comparison with the infection for which the operation was done, the result is certainly far less uncomfortable. I have seen inside the antrums of these patients, and they are clean.

Dr. H. W. Lyman, St. Louis: I have seen a number of patients who were operated on in this manner by an otolaryngologist a short distance from St. Louis, and they were much pleased.

The Newer Conception of Rhino-Otologic Meningitis. Dr. J. Garfield Dwyer, New York.

In twenty-eight years I have personally investigated 367 cases of rhino-otologic meningitis. There were 18 recoveries, apparently not due to any particular procedure. The cell count of the spinal fluid in such cases is no criterion of whether a patient is getting better or not. The bacteriology has been confused, because the staining power of the bacteria varies. Thus, one species may be reported to be two different bacteria, whereas in reality some of the organisms take the characteristic Gram stain while the others, being altered or injured, react

oppositely. A cloudy fluid without organisms does not mean meningitis but is due to an irritant on the outside of the dura. Usually an organism in the spinal fluid means meningitis. A blood count that is persistently high, such as 30,000 to 35,000, without any other symptoms in an adult indicates either irritation of the meninges or pneumonia.

Meningitis may be divided into the fulminating type, the average type, the basilar form and that due to the type III pneumococcus.

Physicians will have to change their whole conception of the pathways of infection of meningitis. Postmortem specimens show that in most of the cases the infection spread from the sphenoid sinus or from osteomyelitis from the nose, and the mastoid operation was simply an end-result. There is no treatment. The means of treatment in the future will be an antiserum.

DISCUSSION

Dr. Ralph A. Fenton, Portland, Ore.: Wide exposure of the dura is safer than pinhole openings. My associates and I have had no trouble if the dura was painted with iodine. We are opposed to tight packing, either in the nose or in the ear, preferring rubber tubes or light cellophane drains.

Safety-Pin in the Esophagus. Dr. Louis H. Clerf, Philadelphia.

This article was published in full in a previous issue of the Archives (**24**:282-288 [Sept.] 1936).

DISCUSSION

Dr. Robin Harris, Jackson, Miss.: I have had 7 patients with open safety-pin in the esophagus, point up. In 3 instances the pin was low and was pushed into the stomach. The pins passed per anum with no trouble or discomfort. Two were removed by changing ends in the stomach.

Carcinoma of the Tongue: A Review of Fourteen Cases. Dr. Frank R. Spencer, Boulder, Colo.

This article was published in full in the Archives, **24**:1-13 (July) 1936.

DISCUSSION

Dr. Thomas E. Carmody, Denver: From the standpoint of irritation, I think that smoking is not so important as many people think. Broken-down teeth, poor dentures and bridgework are much more important. Broder's classification helps remarkably. As to a history of syphilis, I think that one should be careful. Irradiation is not hard to apply if one uses radium seeds. One should make roentgenograms of the chest to see whether metastasis is already taking place. Besides radium, the Coutard treatment, of course, has done much.

Dr. Frederick A. Figi, Rochester, Minn.: Unfortunately, Dr. Spencer has had to deal entirely with late stages. Obviously, the results following treatment in such a group of cases will not compare favorably with those in another group including even a sprinkling of instances of moderately early growth. Biopsy is advisable for diagnosis and grading of the activity of the growth. If a carcinoma of the tongue is well localized, the most satisfactory treatment of the primary growth in my experience and that of my associates at the Mayo Clinic is wide excision by cautery or electrocoagulation, supplemented with implantation of gold radon seeds. If the neoplasm is extensive, especially if it is of a low grade of malignancy and if involvement of the floor of the mouth is not extensive, the entire anterior portion of the tongue may be destroyed by diathermy. Resection of the entire tongue is rarely justified because of the poor prognosis. It sometimes is advisable to treat these lesions by implantation of radium emanation seeds alone, and surprisingly satisfactory results often follow such treatment. In a number of these cases in which the condition originally appeared almost hopeless on examination at the Mayo Clinic, radon seeds were implanted, with the idea of giving the patient some temporary relief. Later, when these patients returned

for observation, the tumors had disappeared. In general, cancer of the tongue and the floor of the mouth is more highly malignant than cancer originating on the lips, cheeks or jaws, and metastasis is prone to occur earlier and more widely. More extensive removal of the cervical lymphatic structures is accordingly advisable. However, in spite of prompt and thorough treatment, the prognosis is as a rule less satisfactory in these cases than in those in which similar lesions appear elsewhere about the mouth. Of 162 patients operated on for lingual cancer at the Mayo Clinic, 156 were traced. Of these, 37.2 per cent were living five years or more after operation. Ninety-nine of these 156 patients presented no evidence of involvement of lymph nodes at the time the treatment was carried out, and 94 of this group were traced; 51.4 per cent of the 94 were living five years or more after operation. With involvement of the regional lymphatics present, however, the prognosis is decidedly changed, the percentage of five year cures dropping to 14.3.

Dr. Sanford M. Withers, Denver: In my cases of buccal cancer, smoking had been practiced for twenty-eight years before the origin of cancer and chewing tobacco for twenty-three years, on the average. In leukoplakia I think that cauterization is unnecessarily severe. Two erythema doses of roentgen rays through the mouth, with filters of 0.5 mm. of copper, will clear up any leukoplakia. If it is localized, a surface application of radium in dental compound will do the same thing. I think that it is wise to do a biopsy in every case of cancer of the tongue. I recommend operation chiefly for those radioresistant malignant growths of the anterior half of the tongue, certainly not, however, for the malignant growths of the base of the tongue. I am forced to the conclusion that most laryngologists are not competent to treat carcinoma of the tongue. I think that they should be, and I wish that more of them were.

Ciliated Nasal Epithelium: Its Culture in Vitro. Dr. Arthur W. Proetz and Marian Pfingsten, St. Louis.

This is a preliminary report of the methods employed and the modest success attained. The cultures grown have shown the practicability of the method for the basic study of this tissue. The tissues employed were fragments of turbinates taken from the guinea-pig fetus at term. The medium employed is essentially that of Vogelaar. We have demonstrated strands of new epithelial cells growing out from the tissue that is being cultured. In some instances typical active cilia are noted. A motion picture was taken with the 16 mm. camera, the single frames being exposed at twenty, forty or sixty second intervals.

DISCUSSION

Dr. Harry L. Baum, Denver: I have no discussion to offer on a presentation of this sort. There is nothing to be said but words of praise.

Dr. Ralph A. Fenton, Portland, Ore.: I should like to ask Dr. Proetz to comment on this: Cultures of tissues from the upper respiratory tract suffer from overgrowth of fibroblasts, perhaps because under present methods there is no provision for excretion from the sample of tissue. Hence, the detritus of broken-down cells is picked up by and activates phagocytic connective tissue cells (histiocytes) which are always found in these tissues and which shortly change into fibroblasts.

A new method of culture of bone marrow recently devised by Osgood and Muscovitz of the University of Oregon (*J. A. M. A.* **106**:1888 [May 30] 1936) provides for circulation and excretory change in the nutrient medium, which reaches the sample of tissue by osmosis through a collodion capsule. Since air also reaches the tissue sample in this method, it offers much hope of continued growth for tissues from the upper respiratory tract and for their experimental subjection to various physical, chemical and medical agents.

Dr. Arthur W. Proetz, St. Louis: Dr. Fenton told me yesterday about Dr. Osgood's work, and I shall look forward to it with a great deal of interest.

TRAGUS TESTS AND FIXATION. DR. EUGENE R. LEWIS, Los Angeles.

It is evident that certain relations have been established; a vibrating fork on the tragus is normally heard louder and longer than one on bone; this relation is also found in obstructive deafness and in nerve deafness (impaired perception), but in fixation deafness this relation is reversed: the fork on the bone is heard after that on the tragus ceases to be heard.

These observations appear to be consistent with what has been outlined concerning tympanic and cochlear mechanics. When both the round window and the oval window are fixed, the perilymph container is blocked at both ends; under such conditions it would appear mechanically impossible for sound waves to produce interaction between the cilia and the tectorium. Either window, if not fixed, may subserve some measure of venting apparently indispensable to tactual stimulation of the end-organs of hearing. In fixation of the oval window, bone-borne effects impressed on perilymph may be effectively vented at the round window, but this would not hold for air-borne or tragus-borne effects; trans-tympanic air-borne waves impinging on the round window could hardly be simultaneously impressed via and vented by the same membrane; relatively little tragus-borne effect is impressed on bone.

DISCUSSION

DR. ISAAC H. JONES, Los Angeles: "How can one see the ear hear?" One can at least observe the motion of the drum membrane and hammer and to this extent can know exactly how well this portion of the ear can hear. The Comprex oscillator with the electric otoscope causes an alternate rarefaction and condensation. One can cause the drum membrane to move about a hundredth of an inch, and such excursions are plainly visible. As to frequency, one can cause the membrane to move three, four, five or any number of times per second. In this sense one is then able to see the ear hear. To me, the best method to determine fixation of the stapes is as follows: If the turning and caloric tests and the audiometric tests in the sound-proof booth reveal a normal internal ear and eighth cranial nerve and the deafness is proved to be conductive and the Comprex oscillator with the electric otoscope shows normal texture and normal mobility of the drum membrane, one can conclude that there is only one diagnosis—one definite lesion—fixation of the stapes.

DR. DAVID HIGBEE, San Diego: I was particularly glad to see Dr. Lewis demonstrating hearing taking place when the stapes was fixed. Otologists all know that hearing does occur when the stapes is fixed, and that eliminates one of the ideas which have been taught—that the stapes must go in and the round window out. Experimental work which has been done proves that hearing occurs when the stapes is fixed and the round window niche is filled with cementum. Under those circumstances, according to the theories advanced, there could be no hearing whatever because the round window does not bulge, and it would be necessary that there be some release of the pressure built up there in order for hearing to occur, and if that pressure could not be released, there could be no hearing. I think that this is a demonstration of the fact that the vascular bed and the saccus endolymphaticus are sufficiently resilient to take up that pressure and transmit it and that although the labyrinth is a solid body, there are other ways for pressure to escape than through the round window.

DR. EUGENE R. LEWIS, Los Angeles: I believe that in the instances mentioned the round window membrane was only weighted, rather than actually fixed.

A METHOD OF STUDY OF THE NASAL MUCOSA IN RELATION TO THE TUBERCLE BACILLUS. DR. STEPHEN A. SCHUSTER, El Paso.

This method of epithelial scraping offers certain advantages in the study of nasal bacteriology in general and is of particular value in showing the existence and incidence of tubercle bacilli in the mucosa of the tuberculous.

Of the fifty unselected patients with pulmonary tuberculosis, 30 per cent showed tubercle bacilli in the epithelial scrapings, but in the smears of material from the noses of these same patients no organisms were found.

Eighty-six per cent of the patients showing tubercle bacilli in the nasal mucosa had pathologic deviation of the nasal septum.

The data on age and sex incidence were inconclusive.

Entrance of tubercle bacilli into the nasal mucous membrane may be an ectogenous invasion, caused by fingers, handkerchief or bacilliferous sputum, or an endogenous invasion, by way of the blood vessels and lymphatics.

Patients showing tubercle bacilli in the nasal mucosa may possibly be considered as having potential nasal tuberculosis.

Since 28 per cent of the patients with bacilli in the mucosa yielded variation or granular forms of the organisms but only 16 per cent yielded rod forms, since the variation forms are avirulent or dead organisms and since the latter forms are found when the lytic action or the resistance toward the organism is great, one may possibly conclude that in many cases primary nasal tuberculosis is caused by avirulent or dead tubercle bacilli or their chemical products.

This method of epithelial scraping may be of value in studying the nasal bacteriology in different acute and chronic infections of the mucosa. Since the bacterial flora of the nose is so complex and since it is difficult to determine which organism is the true infective agent, it is correct to conclude that the organism or organisms found subepithelially are probably the predominating organisms in the disease, and more than likely are the causative factors.

DISCUSSION

Dr. Robert Levy, Denver: This study by Dr. Schuster calls to mind the work of George Wood and also that of Walshon. One may be justified in speaking of clinical tuberculosis and pathologic tuberculosis. It has been possible to demonstrate tubercle bacilli in scrapings but not often enough to make it dependable for diagnosis. The point I wish to make is that in all the cases of suspected tuberculosis of the nose the diagnosis cannot be proved by any one of these methods and that eventually experimentation with animals is absolutely necessary. By and large one can often arrive by exclusion at a reasonably sure diagnosis of tuberculosis of the nose from the clinical findings.

Dr. W. W. Pearson, Des Moines, Iowa: Dr. Schuster's cases lead me to believe that the bacilli were of endogenous origin, brought to the nose in all probability through the circulating blood. This hematogenous dissemination demonstrates the importance of a thorough conception of disease as shown by the pathologic picture indicated by dead and living tissue.

The Aeration of the Nasal Accessory Sinuses After Birth. Dr. W. W. Wasson, Denver.

A series of slides showing the development of the nasal accessory sinuses are presented.

DISCUSSION

Dr. Voss Harrell, Detroit: Dr. Wasson's report of his excellent research more or less limits the discussion by a clinician to one of commendation and appreciation.

Great difficulty was encountered in Detroit in finding any additional knowledge. Not only the pediatricians and the rhinologists but the roentgenologists confess their lack of knowledge and evidence in the understanding of these infantile sinuses.

One may conclude, however, that severe infantile nasal infections will retard the development, and clinically Dr. Shea has shown that such dwarfed sinuses have a great tendency to show chronic sinusitis.

Physicians also now know that infantile sinusitis is not eradicated until one can show that pneumatization is reestablished.

It is now necessary for roentgenologists to show when and how one may determine this. I believe that many physicians have greatly neglected to make roentgen studies of these infantile sinuses. This work I am sure will stimulate those present to request roentgen studies in all instances of infantile sinuses.

Book Reviews

Plastic Surgery of the Nose. By J. Eastman Sheehan, M.D. Second edition. Price, $9.00. Pp. 177, with 131 illustrations. New York: Paul B. Hoeber, Inc., 1936.

Sheehan's book on nasal plastic surgery was probably the first of its kind to appear in this country in the postwar period and was received with great interest. Eleven years have elapsed, in which a number of works on the same subject have been published by other authors. The second edition of Sheehan's book finds him still in the van. Some of the older technic has been discarded, and new angles have been introduced as a result of accumulated experience, so that one notes a tendency to conciseness and clarity in the new edition, which makes it more easily followed. As in the first edition, stress is laid on the fundamentals and much of the detail of preoperative and postoperative care is discussed in a practical manner. The chapters on the operation on the septum, transplantation of costal cartilage, the syphilitic nose and skin grafts are particularly well done.

In all it is a book that every rhinologist will find profitable to read whether he is especially interested in plastic surgery or not.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko-Kirurgiska Institutet, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143 Groningen, The Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Robert F. Ridpath, 1737 Chestnut St., Philadelphia.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Atlantic City, N. J. Time: June 7-11, 1937.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Detroit.

American Bronchoscopic Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

American Laryngological Association

President: Dr. W. B. Chamberlain, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Hotel Traymore, Atlantic City, N. J. Time: June 3, 4 and 5, 1937.

SECTIONS:

Eastern.—Chairman: Dr. D. C. Jarvis, Quarry Bank Bldg., Barre, Vt.
Southern.—Chairman: Dr. Charles D. Blassingame, P. & S. Bldg., Memphis, Tenn.
Middle.—Chairman: Dr. Alfred Lewy, 25 E. Washington St., Chicago.
Mid-Western: Dr. Frederick A. Figi, 436 Tenth Ave., Rochester, Minn.
Western.—Chairman: Dr. David R. Higbee, 3245 Fourth Ave., San Diego, Calif.

American Otological Society

President: Dr. Edmund Prince Fowler, 140 E. 54th St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.